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PNEUMONOKONIOSIS IN NEW SOUTH WALES COAL MINERS.

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As long ago as 1907, Summers, of Bendigo, Australia, first pointed out the value of X-ray examination of the lungs in the diagnosis of pulmonary dust disease.

THUS stated Dr. Charles L. Sutherland in 1945, and from this beginning Australia has not lagged behind. The excellent work of Badham and Tyler, the report of 1923 of the Broken Hill Commission (Chapman, Smith and Edwards) and the subsequent industrial legislation have shown that the medical fraternity has been wide awake to the "dust hazard".

Examination of a person suspected to be suffering from pneumonokoniosis should be undertaken by a group of physicians knowledgeable and interested in this type of medical diagnosis. After all, the number of medical men who have the opportunity of studying any form of *pneumonokoniosis is small indeed and the number of* those people who are running the risk of this type of infection is also small, when classified as a percentage of the total population.

The desirability of one special board or branch to deal with all dust cases, whether they are caused by gold, quartz, coal, tin *et cetera*, has so far not been recognized and at present there are three separate boards operating in New South Wales.

There are even two separate boards dealing with the coal-mining industry, only because the miners of the South Coast decided to disagree with the medical decisions based on long experience of the original board. The other two large coal-mining districts, north and west, showed their appreciation of these years of investigation and subsequent knowledge obtained by saying they preferred to have their men examined and the compensation determined by those who had for so long taken such a medical interest in the effects of dust in coal miners.

This "old" board (the New South Wales Special *Pneumonokoniosis Board*) has been in existence since 1932 and has examined as many as 236 coal miners in one year. The constituting of the board was the result of a survey of South Coast miners (with X-ray films taken at Waterfall Sanatorium). Dr. Charles Badham induced the *government of the day to appoint* this small group thoroughly to examine the whole situation and the amount of distress that might be caused by coal dust. The group of medical men appointed, Dr. C. Badham, Dr. Cotter Harvey and Dr. H. M. Cutler (as radiologist), apparently constituted the first scientific body to deal with pneumonokoniosis relating to coal miners.

The Board.

This "Special Pneumonokoniosis Board of New South Wales" now consists of a chairman, who, quite apart from carefully assessing the value of each other member's contribution to the final medical opinion, undertakes the duty of previously interviewing the applicant, of taking full notes of the man's history and, in particular, of *carefully annotating the number of years (even to* months) that have been worked in various mines. This history-taking is of the utmost value and, as will be shown later, the period that has been worked in each particular mine, whether it is in Australia or overseas, is of extreme importance to the examining body as a whole. Apart from the chairman, the board consists of the medical officer of industrial hygiene, two senior physicians and a *radiologist. The only comment that may* be made is that the presence of two physicians at times appears somewhat redundant, if one classes the industrial officer as having sufficient clinical acumen to warrant his appointment and thus make a third consultant physician.

The radiologist, also, as in his daily work, is the equivalent of a spare physician on this board. His fluoroscopic and radiographic evidence, combined with clinical investigations carried out in conjunction with his fellow members, considerably assists in the final summing up of the case.

This board still appears somewhat unique. America has suggested something similar, but with still more medical personnel. England does much work through various committees which come and go. South Africa has a research organization, but does not appear to deal with the economic and social questions so intimately.

The Examination of the Patient.

In an attempt to demonstrate how thoroughly these suspected "dust cases" are investigated and how seriously these investigations are undertaken, the following is a résumé of the "Special Pneumonokoniosis Board" proceedings.

Some days previously a form is lodged by the secretary of the miners' lodge for the examination of a man. An appointment is given to that man to appear at a certain date. His train fare (first class) and his expenses are paid. He arrives in Sydney, where he is immediately sent to the board's radiologist. Stereoscopic films of the thorax and also films of the nasal accessory sinuses are prepared. Before the man is allowed to leave the radiologist's laboratory a fluoroscopic (or screen) examination is made. This work is undertaken to determine: (i) the amount of excursion of each side of the diaphragm (and at the same time to assess as far as it is possible the extent of costal muscular exertion in causing air entry into each lung); (ii) any fixation of the pleura, particularly at each costophrenic angle; (iii) the amount of air entry into each lung field which is of all things so important; (iv) any opacity of the apex or elsewhere of each lung; (v) the size of the heart and great vessels leading therefrom and any departure from normal outline; (vi) any opacity of the posterior portion of the mediastinum, the fact being remembered that the left ventricle of the heart, if apart from normal, pushes itself into this space. Enlarged hilar and mediastinal glands may make this space opaque.

The films are processed and dried and are ready for discussion by all members of the board within two hours of the man's arrival in the X-ray laboratory.

For some months, in addition to the stereoscopic films of the thorax, extra films were made during deep inspiration and expiration. Two hundred of these measurements relating to the extreme movements of the diaphragm were collected. The results of this investigation will be set out later.

Our coal miner, having for the time being finished with the radiologist, reports to the chairman of the board, where a careful record is taken of his industrial history. It is from this history that one learns of the country and district in which is situated each mine in which he has worked.

Careful notes of the man's industrial history must be carefully studied. Experience has shown that different districts, whether they are in Australia or overseas, show variations in the fibrosis (nodular or linear) that the lung fields may exhibit. Mines in the same area situated only a short distance apart, on account of the change in the silica content of the coal seam or in the less or greater amount of quartz present, are known to modify the pictures of the lung tissues considerably.

When the members of the board meet in the afternoon the man is stripped, his weight and height are determined, and the full medical examination is carried out. This includes the urine test, estimation of blood pressure, and so on. The examination of each man is in fact comparable with that of an applicant for a life insurance policy, except that the miner has X-ray films prepared of his chest and sinuses, and five medical men, instead of one, to determine his physical fitness. The man whose condition is being investigated may produce any certificates, historical records or any X-ray films from outside medical sources. These are all carefully looked at, compared, and marked as having been examined. Pulmonary and cardiac sounds are investigated, whilst in the background there is the stereoscope displaying films of the chest. The radiologist in person demonstrates the abnormalities, if any, that he may see on these stereoscopic films and, in addition, uses a stethoscope himself in an attempt to compare the clinical with the radiographic findings.

Over the years the New South Wales Pneumonokoniosis Board has insisted on first-class stereoscopic examination. With this process, when eyes are accommodated, the thorax is seen as a complete barrel; the ribs are seen from their junction with the spine to the cartilaginous synchondroses; the heart floats in the centre; the lung markings are depicted and the peribronchial and perivascular fibrosis is clearly delineated. The nodules at the antra of the alveoli are clearly seen, and it is thus felt that the amount of infection can be estimated.

Very occasionally serial films taken at regular periods over years are available. The just evaluation regarding the amount of pneumonokoniosis present is made much simpler. Sometimes films of the survey carried out at Waterfall Sanatorium in 1932 may be obtained. Here any increase in nodular and linear fibrosis that has occurred over the years is very evident. Men whose disability some years ago was considered by the board to be little or none often reappear for examination. Here again previous films which have been carefully filed by the commission are brought forward and the information gained from comparison is invaluable. Incidentally, the accurate diagnosis of any pulmonary condition is enhanced if serial radiographs are carefully studied. This applies more to early tuberculosis probably than to other lesions, and the problem of active or quiescent disease can really be gauged only by serial examination.

Quality of films is of importance. Despite the modern high-powered X-ray plants with an almost foolproof interlocking mechanism, despite technicians who have been carefully trained over many months or even years, despite the commercial firms' interest to see that their apparatus turns out the best technical results, the human element comes into the picture to such a great degree that comparison between films taken at different institutions and in different laboratories is at times out of the question. This fine reticulosis, these nodules, so small and almost transparent, need the best technique and most careful dark-room procedure to allow a correct estimate of the disease to be put forward. It is for this reason that the board decided many years ago that all applicants for examination should be sent to their own radiologist and that all necessary examinations should be undertaken by that man alone. The result has been that for those miners who after years may reapply there are films and reports of comparable value.

Lately, in an attempt to assess the amount that bronchitis has to play in the disability of these miners, X-ray films of nasal accessory sinuses are also prepared.

After the ordinary clinical examination is complete the man is then exercised, if his general condition will allow this to be undertaken.

Many men are not asked to undertake the exercise tolerance test because the clinical examination has already determined that the amount of nodular fibrosis (pneumonokoniosis) in each lung field or, far more often, the cardiovascular degeneration is such that extreme exertion, even for a short period, is unwarranted and inadvisable. If, however, no deleterious effects can be expected from exercise, the man is asked to "touch his toes" twenty times as rapidly as possible. This appears a satisfactory test. The respiratory and pulse rates are noted beforehand and, the exercise being completed, respiration and pulse rates are again recorded at minute intervals, to determine if these return to within the original limits. After five minutes all readings are duly recorded with other clinical investigations. The medical board then attempts to arrive at a decision regarding the man's fitness for work.

This "exercise tolerance" test seems somewhat primitive, but so far it has been the most satisfactory. Other exercises that have been tried are lifting heavy weights a number of times, walking up flights of stairs, and

stepping on and off a chair. For a period, tests with a spirometer were instituted but were discarded as being unsatisfactory.

All data having been written down, the five medical men discuss all that has been found. It is here that the radiologist can not only assist the other members of the board, but can himself learn much. Crepitations at the base of the right lung can be compared with the amount of linear fibrosis in that area on the X-ray film. If "cardiac insufficiency" is found clinically, it may be demonstrated radiologically by the amount of œdema at the base of each lung, and so on.

The screen examination findings are also taken into account. The degrees of nodular fibrosis seen on the X-ray films have been arbitrarily fixed at (i) very early, (ii) early, (iii) moderate, (iv) well marked, (v) advanced. With these defined stages the amount of linear fibrosis in each lung consistent with age must be borne in mind and the two types of fibrosis considered together. A very early degree of nodular fibrosis cannot, it is felt, by itself warrant taking a man off full work. The amount of linear fibrosis that may be present cannot, it is at present considered, be due solely to working in the coal industry. After all, the same increase in linear markings may be found in members of the civil service or in the man who has returned from New Guinea or from any other theatre of war.

It is not till after the miner has left the room that the medical referees arrive at the amount of disability present and the man's fitness for work. Sometimes the discussion is lengthy and different factors affecting the man's condition are debated. The miner may have no evidence of pneumonokoniosis but still be totally unfit for many reasons—for instance, on account of an advanced cardiac lesion. So it may come about that no compensation is awarded but the applicant is marked totally unfit for work.

So it comes to this, that no medical board dealing with cases of industrial hazards is complete without a radiologist well trained and competent to discuss the differential diagnoses that may occur.

Discussion.

From nearly fifteen years' experience as radiologist to this special pneumonokoniosis board an attempt is now made to evaluate in some degree the cause of fibrosis of the lung tissue, the reasons for radiological diagnosis and the effects of dust on the coal miners in New South Wales.

During the 1914-1918 war large numbers of men were affected by gas. A radiologist who has followed up the effect of gas on the lungs of these soldiers realizes the effect of this gas as depicted on an X-ray film. It is definite and distinguishable from any other form of pulmonary fibrosis. This irritation effect of gas is a picture of fine Honiton lace—a fine reticulosis—and is one of the many pictures of fibrosis seen radiologically. This is mentioned only to demonstrate that the types of linear fibrosis may be so varied both in causation and in radiological appearance.

The amount of increase in linear fibrosis due to chronic inflammation of the bronchial lining in coal miners is not in any way greater than that found in the ordinary community. This fact was particularly brought out after an examination of thousands of X-ray films of returned soldiers during the last two or three years. If, then, a coal miner does not suffer from chronic bronchitis any more than anyone else, it seems that this condition should not be considered when compensation in relation to his work is assessed. On the other hand, the industrial nodular fibrosis, the reticulosis of Great Britain, is of great importance and must always be taken into account. Immediately the question arises whether these different types of linear fibrosis can be differentiated on an X-ray film. Of this there is no doubt. The distribution alone of the fibrosis due to chronic bronchitis will allow the competent radiologist to recognize it. The finer reticulosis type again occurs only in pulmonary tissues which have been damaged by some form of dust and it is not seen in the civilian workers.

Pathologists make the statement that the only place in the lungs of silicotic patients in which connective tissue grows is within the lumen of the blood vessel.

It seems, therefore, that it would be better to forget the ordinary peribronchial linear fibrosis in determining the amount of damage caused by dust, or to report on the presence thereof just as one would for the army or in a mass survey. The true reticular or connective tissue type of fibrosis would only then be taken into account with the realization that this connective tissue is that which grows actually within the lumen of the blood vessel. It is this marking on the radiograph that is the cause of the dyspnœa.

The advisory committee to the Medical Research Council of Great Britain classes the coal fibrosis of South Wales as one of reticulosis. This reticulosis is recognized by the pneumonokoniosis board as a definite entity, but the appearance of the X-ray films of those who have worked solely in New South Wales is extremely different, no matter what the degree of anthracosis in each case. When a combination of irritating factors occurs it becomes still more difficult to assess the amount of damage caused by silica, whether in Australia or in other parts of the world, and almost impossible to recognize the proportionate percentage of damage caused by the dust of each particular district.

Reticulosis somewhat similar to that found in the Welsh workers is more common in the miners who have worked in the northern and western fields, where the damage to lung tissue, clinically, radiologically and pathologically, is so much less than in other areas.

After miners have arrived from the Welsh, English or Scottish coalfields and then worked for some years in the mines of New South Wales, these fine, rather soft or translucent nodules become larger and denser—more opaque—to X radiation. The original underlying reticulosis, however, remains and must be taken into consideration when the amount of disability is considered. Radiological appearances in overseas workers suggest that the silica content or the chemical action of the silica in British mines may have a different effect on the lung tissue from that of the dust inhaled by New South Wales miners. There also appears to be some difference in lungs of the men who have worked in the northern, western or southern fields.

It is unfortunate that the two different terms "reticulosis" and "nodular fibrosis" are used by separate authorities to mean the same thing—that is to say, anthracosis.

On the other hand, although the cause and the resultant injury to the pulmonary tissue is the same, there is undoubtedly a variation in the appearance on the X-ray films. The British films show somewhat more linear distribution; the nodules appear smaller and closer together. However, the origin is the same and is really only more delicate in structure. Therefore it seems wise that nodular fibrosis as a descriptive term should be retained by the New South Wales board.

No matter how acceptable various theories may be, it is to be realized that silica is the cause of the disability in coal miners as well as in other miners. It should also be realized that, other things being equal, the miners working in a pit with the greater content of silica in the coal or seam, and therefore in the dust, will show the greater percentage of cases of lung disease.

It has been said that a coal miner who has had bronchitis for many years is very unlikely to become "dusted". The men from Wales and Cumberland and other British mines have grown from childhood with a certain amount of intercurrent pulmonary infection. As a result, they continually have a large amount of sputum which has to be excreted; so that any dust inhaled has less chance, on account of this mucous excretion of the bronchioles, to irritate the lining of the pulmonary airways.

The necessity for X-ray examination of all people discharged from the services who are applying for a pension has allowed a large amount of instructive information to be collected.

This information is not only for immediate but for future reference. As can be imagined, the report given on the great majority of these films is: "Fibrosis within normal limits". This statement, however, is really one of comparison, for the man or woman who has been overseas, whether in New Guinea or the Middle East, has different thoracic X-ray findings from those of the normal civilian.

TABLE I.

Details of Findings of Pneumonokoniosis Board in the Examination of Miners. Data are distributed according to age groups and to coalfields in which the miners were employed for the five-year period ended June 30, 1946.

Age Group. (Years.)	Total Number of Miners Examined.	Distribution of Miners by Examination Findings.			
		No Evidence of Pneumonokoniosis.	Evidence of Pulmonary Fibrosis Due to Dust.		
			Non-Incapacitating	Partially Incapacitating	Totally Incapacitating
(a) Northern Coalfield, New South Wales:					
40 and under	5	4	1	0	0
41 to 50 ..	15	8	1	4	2
51 to 60 ..	73	38	9	13	13
61 and over	9	4	1	2	2
All ages ..	102	54	12	19	17
(b) Southern Coalfield, New South Wales:					
40 and under	26[1]	9	7	8	2
41 to 50 ..	49	7	11	21	10
51 to 60 ..	116	28	17	37	34
61 and over	27	5	5	10	7
All ages ..	218	49	40	76	53
(c) Western Coalfield, New South Wales:					
40 and under	0	0	0	0	0
41 to 50 ..	19	4	5	7	3
51 to 60 ..	33	8	3	10	12
61 and over	6	2	0	2	2
All ages ..	58	14	8	19	17
(d) Two or More Coalfields in New South Wales:					
40 and under	0	0	0	0	0
41 to 50 ..	6	2	2	2	0
51 to 60 ..	26	7	3	12	4
61 and over	6	2	0	1	3
All ages ..	38	11	5	15	7
(e) Coalfields in New South Wales and in Other Australian States or New Zealand:					
40 and under	0	0	0	0	0
41 to 50 ..	1	1	0	0	0
51 to 60 ..	9	4	2	2	1
61 and over	6	1	2	2	1
All ages ..	16	6	4	4	2
(f) New South Wales and British or American Coalfields:					
40 and under	5	2	0	3	0
41 to 50 ..	57	19	12	16	10
51 to 60 ..	174	44	24	60	46
61 and over	40	5	6	14	15
All ages ..	276	70	42	93	71

[1] Includes three men aged thirty years or less, two of whom showed no evidence of pneumonokoniosis and one of whom was partially incapacitated by pulmonary fibrosis due to dust.

TABLE I.—*Continued.*

Details of Findings of Pneumonokoniosis Board in the Examination of Miners Data are distributed according to age groups and to coalfields in which the miner were employed for the five-year period ended June 30, 1946.—Continued.

Age Group. (Years.)	Total Number of Miners Examined.	Distribution of Miners by Examination Findings.			
		No Evidence of Pneumonokoniosis.	Evidence of Pulmonary Fibrosis Due to Dust.		
			Non-Incapacitating.	Partially Incapacitating.	Totally Incapacitating.
(g) Collieries in New South Wales as well as in Other Mining Industries:					
40 and under	1	1	0	0	0
41 to 50 ..	9	4	1	4	0
51 to 60 ..	40	8	5	8	19
61 and over	8	0	1	4	3
All ages ..	58	13	7	16	22
(h) All Places of Employment:					
40 and under	37[1]	16	8	11	2
41 to 50 ..	156	45	32	54	25
51 to 60 ..	471	137	63	142	129
61 and over	102	19	15	35	33
All ages ..	766	217	118	242	189

[1] Includes three men (all from the southern field) aged thirty years or less, two of whom showed no evidence of pneumonokoniosis and one of whom was partially incapacitated by pulmonary fibrosis due to dust.

Geographical regions—perhaps continual humidity—may have some effect on the increase in the amount of linear markings seen on an X-ray film of the returned soldier. So pronounced is this feature that, in order to confirm my own opinion, a number of films of these ex-service people have been shown to other radiologists, to members of the New South Wales Board of Health and of the Industrial Hygiene Department, and to physicians interested in the question of chest pathology. In these returned men there is a reticulosis very much coarser and more delineated (spread out) than that mentioned previously in the gas subjects of 1914-1918, and even more pronounced than that found as an early reticulosis by the Medical Research Council in the miners of South Wales.

This increase of linear markings, as long as it is within quite broad limits, apparently causes no disability, no clinical signs, no departure from any athletic activity that the normal Australian may undertake. Under these circumstances it does not appear right to label all these returned men with an insidious diagnosis purely radiological in origin. Whether years later some disability may be caused or the lung fields remain the same without any resultant complication or whether the fibrosis to a great extent disappears remains to be seen.

The type and distribution of lung markings in men working in hazardous occupations varies considerably. Briefly, dust with the greatest silica content is the dust of greatest evil.

The quartz miner may contract silicosis in five to seven years, the metal worker without proper precautions in under ten years; the asbestos worker, the flour miller and others take longer still. The soft coal miner, and only "soft coal" is found in Australia, must work at least twenty years before manifestations of anthracosis occur.

The distribution of the nodules again varies considerably. In the worker in quartz the mid-zone of each lung first shows evidence of dust invasion, and the amount is similar in quantity on each side. This mid-zone invasion occurs also in the coal miner, but not quite so definitely, as the nodules tend to spread a little more, both upwards and downwards. In workers in asbestos, in flour and even in metal the distribution of the nodules is far more general throughout both lungs.

The above phenomena are quite easily discernible under the lower magnifications of a microscope and can be demonstrated on a slide with a thin section of pneumonokoniotic lung floating in saline solution. Radiologically, the demonstration of emphysema in a lung in a living subject has not been considered altogether satisfactory. Of late years, however, the degree of emphysema may be assessed more by fluoroscopic examination than by other radiological means. When the chest is being screened a very low voltage is passed through the tube, so that the differentiation between the soft tissues may be more easily considered. Preliminary accommodation of the eyes is, of course, all-important. In Vienna, when I was last there, the country was too poverty-stricken to afford X-ray films, so that with a Viennese instructor (who had an Oxford accent) thirty-five minutes in the dark were demanded before an examination of any chest was attempted. When emphysema is present it is denoted by the extraordinary translucency of the lung tissue and, even more than this, by the lack of variation in translucency on expiration or inspiration. This appearance usually occurs at the thickest portion of the lung—the base. The air intake of these patients must be very small and the fibrous walls between the alveoli to a great extent destroyed. Emphysema on the X-ray film greatly masks any linear or nodular fibrosis and, therefore, should all the more be taken into account in assessing the disability due to dust.

Emphysema, however, occurs in any portion of the lung. It is found more at the base of each lung, on account of the difficulty of expiration when small nodules act as a ball and socket valve in these lower lung areas. Large emphysematous bullæ may at times be found towards the apex as well as towards the base of any anthracotic lung.

The action of the fibrotic nodule at the junction where the smallest bronchioles enter the alveoli is that of a ball and socket pump. The air may be taken into the alveolus, but the nodule at the outlet prevents the air from being expired. The next act of inspiration may allow a still further small amount of air to enter the sac, until eventually the wall becomes more and more stretched. With a similar process taking place in the adjacent alveoli the intervening walls break and allow intercommunication. With these air cells breaking one into the other, it is quite simple to realize how, as the process extends, large areas of usually available epithelium are lost and the intake of oxygen is much diminished on account of the diminished surface area.

It may be definitely recognized that emphysema occurs only when fibrosis is present. On the X-ray films pneumonokoniotic bullæ are often seen as large and small cavities throughout the lung tissue. These cavities do not have the opaque edge that is common in a tuberculous subject, but fade into the surrounding parenchyma, with the result that they are much more difficult to demonstrate. At post-mortem examination it is found that these bullæ are caused by the breaking down of the alveolar walls as a result of the block to the exit of air by the fibrotic nodule. As more dividing walls collapse, so do the bullæ grow larger and larger.

Measurements related to the movements of each cupola of the diaphragm have been sought, both by fluoroscopic methods and also by atempting to gauge the level of the diaphragm on films taken in expiration and inspiration. This has not been easy. The cooperation of the patient is not altogether satisfactory and, even if this cooperation is given, it is difficult at times to make the man understand what is wanted.

The following conclusions have been reached.

1. The maximum excursion of either side of the diaphragm in any miner examined has been five centimetres.

2. One side of the diaphragm may not move at all whilst the excursion of the other side may amount in the same man to only 0·5 to 0·75 centimetre. In these cases it will be found that the chest expansion reaches at least 5·0 to 7·5 centimetres. The air intake is thus muscularly contributed by the intercostal and other extrathoracic muscles as well as by the diaphragm.

The difference in elevation of the diaphragm does not entirely depend on the nodular fibrosis present. The amount of thickened pleura at each costo-phrenic angle, along the interlobar fissures and at other points, such as at the level of the second rib on each side, must be taken into account. To this thickening of the pleura must be added the increase in linear fibrosis throughout each lung, some of which may be consistent with age or due to long-standing bronchitic changes.

When a pronounced degree of emphysema is present, particularly when this occurs at the bases of the lungs, the amount of air intake is much lessened and the movements of the diaphragm are restricted.

In chronic bronchitis the fibrosis is much more coarse and is found towards the hilar and mediastinal glands. Again, although overseas authorities mention enlargement of these hilar glands in coal miners, no gross increase in size is noticeable in the New South Wales miners.

Before I pass on to the appearances in New South Wales a comparison with the X-ray findings found in English workers may be considered.

1. The "reticulosis", so-called, occurs about the same period as the "very early" or "early" nodular fibrosis is found in this State. In addition to this reticulosis, as part and parcel of it, there are seen the earliest appearances of nodular marking.

2. Hilar and mediastinal glands are more decidedly enlarged. There is practically no alteration in their size and shape in Australian miners.

3. Old tuberculous, probably healed, particles are less frequently seen in the Australian worker. In fact, it is uncommon to find any evidence of tuberculosis, active or healed.

TABLE II.

Distribution of Tuberculosis, According to Medical Board's Findings Among Coal Miners Who have Worked Exclusively in New South Wales Coal Mines, for the Five-year Period Ended June 30, 1946.

Coalfield.	Number of Miners Examined.	Number of Cases of Tuberculosis.		Percentage of Tuberculosis Among Men Examined.	
		Doubtful.	Certain.	Doubtful.	Certain.
Northern ..	110	2	2	1·8	1·8
Southern ..	243	2	2	0·8	0·8
Western.. ..	63	0	1	0·0	1·6
All fields ..	416	4	5	1·0	1·2

The men of the northern fields, where free silica is less, may show a very early but somewhat similar condition to that described in English and Welsh surveys.

It is only after an examination of a large number of radiographic films of men from England and comparison of the markings with those of the northern collieries that this conclusion has been reached.

The South Coast silica content is higher. Workers present a far more difficult problem. The coal is "harder", although even in these mines it does not approach the anthracite coal of Wales. Nodular fibrosis is undoubtedly more prevalent and the concurrent conditions of bronchitis and cardiac degeneration appear more common. If we deal first of all with the actual infiltration of dust into the lung, this appears much more typically "nodular". The amount of disability due to coal dust can, if no other complications are present, be straightforwardly determined.

Two other complaints cause much worry and make these miners wholly or partially unfit for work. The first is the amount of linear fibrosis, which is often excessive and cannot necessarily be attributed to working in coal. It has been found throughout examinations carried out over the last fifteen years that, if much linear fibrosis due to old bronchitis is present, then there are found little, if any, signs of nodular infiltration.

It can quite well be imagined that, if a man has had bronchitis for years and has been expectorating a moist sputum, the silica particles have not had any chance to settle in the bronchioles, but have been forcibly ejected in the secretion.

Despite the great increase in linear fibrosis that is found in these lungs, although the examinee may complain of disturbed rest at night on account of coughing, it is seldom that a man will cough whilst he is under examination, which usually takes forty minutes or more. Again, even with the large amount of peribronchial and perivascular markings, the stethoscope seldom picks up any moist sounds, and then usually at the bases only. The examinee will not even cough after having performed the required exercise tolerance test of touching the toes twenty times in rapid succession. This has made one think of other causative agents for the linear fibrosis. It may be that a connective tissue lining forms within the bronchioles, destroying the ciliated epithelium, and gives rise to this marked breathlessness, which is shown by radiological examination to be the result of linear and not nodular fibrosis. May this then be the result of inhalation of dust?

If this is so, the problem then arises as to how we are to differentiate between the linear fibrosis due to dust and the linear fibrosis found in workers away from dust, when, in the latter, fibrosis may be due to healed bronchitis in childhood or to still active chronic inflammation of the air passages. So many people who have not seen a coal mine have a similar fine linear fibrosis throughout their lung fields. It appears that, in this country at least, some nodular fibrosis, however slight, must be seen before it can be considered that a man's lung has been contaminated with dust.

Only extensive research with post-mortem examination of lungs of all classes of workers can decide this question. The other pronounced disability that is found in these men of the South Coast—85% of those examined are fifty years of age or more—is cardio-vascular disease. An applicant may have sufficient nodular fibrosis to warrant assessment of, say, a disability of from 33⅓% to 50% and, in addition, he is found to have a cardio-vascular lesion causing 100% disability. He is marked "unfit", but, as can be imagined, it is difficult to explain to the man's colleagues, or to the man himself, that he is completely unable to work, although he receives only a third or half of the possible compensation permissible. This is one of the main causes of the miners' dissatisfaction with the examining medical board. Still, under the present act, the Special Pneumonokoniosis Board is asked only to judge as closely as possible the amount of incapacity that has occurred as a result of the man's work in coal mines. Again, the city clerk or auctioneer may develop a "heart condition" which has nothing to do with any work in coal mines.

One other factor that appears to have little effect on the man's disability is his general physique.

The small, almost undernourished miner with evidence of rickets in childhood appears to be able to carry on throughout his life this heavy, arduous, strenuous and uncomfortable job of coal mining, often just as well as the tall, large-framed, muscular individual who has never been off work in his life.

Again, it is to be stressed, when talking of coal as being a soft dust, that the seams in New South Wales in no way approach the anthracite (hard coal) of South Wales. The soft coal of New South Wales may not have the calorific value of that of Wales, but it certainly does not appear to cause the same amount of irritation of the lung tissue. On the other hand, it must be recognized that this anthracite coal causes very little damage compared with the quartz in which the sandstone miner works.

The future of a coal miner with anthracosis is a vital problem. Despite the more cheerful prognostications to the contrary which have been put forward by Welsh and American investigators, it appears that, once the lung is "dusted", transferring the man to an outdoor or any other type of occupation has not the slightest effect on the progress of the disease. Nodular fibrosis goes on gradually increasing and extending further and further towards the periphery and to the apices. The nodules so small and discrete at first become larger, and bullæ not previously seen make their appearance. It was found by the Committee on Industrial Pulmonary Disease (Medical Research Council), 1945, that, at autopsy, in the lungs of anthracite colliers there was no significant increase in dust (total silica or quartz) with increasing degrees of fibrosis.

Cole[2] states that, when the blood vessels and bronchi are cut transversely, dust-laden phagocytes appear as a ring adjacent to the muscular coat. This condition occurs when there is a moderate amount of dust containing a low percentage of silica. It is the subjects with this condition who rarely develop symptoms until the later decades of life.

Table III.

Details of Findings of Pneumonokoniosis Board.
Data are distributed according to coalfields in which the miners were employed for the five-year period ended June 30, 1946.

Coalfields where Employed.	Total Number of Miners Examined.	Distribution of Miners by Examination Findings.			
		No Evidence of Pneumonokoniosis.	Evidence of Pulmonary Fibrosis Due to Dust.		
			Non-Incapacitating.	Incapacitating.	
				Partially.	Totally.
Northern Coalfield, New South Wales	102	54	12	19	17
Southern Coalfield, New South Wales	218	49	40	76	53
Western Coalfield, New South Wales	58	14	8	19	17
Two or more coalfields in New South Wales.. ..	38	11	5	15	7
Coalfields in New South Wales and in other Australian States, or New Zealand ..	16	6	4	4	2
New South Wales and British or American coalfields	276	70	42	93	71
Coalfields in New South Wales as well as in other mining industries (including shale) ..	58	13	7	16	22
Total ..	766	217	118	242	189

The life of a miner working in coal is much longer than the lives of those working in mines (gold *et cetera*) which contain a greater percentage of silica. Coal dust (apart from the small silica content) is a soft dust, with the result that the linings of the bronchioles are slowly covered with a protective medium. As the late Charles Badham facetiously remarked: "coal dust leaves very little room for silica and even less for a tuberculosis germ to park". The incidence among coal miners (in New South Wales) of superadded tuberculosis appears far less than that among the remainder of the population. In coal miners it is 1 per 1000, whereas for the whole population it rises to 3 per 1000. Coal dust does, therefore, apparently act in some degree as a preventive against tuberculosis. This is completely opposed to the occurrence of quickly added phthisical lesions in those who have contracted silicosis from quartz mining. The quartz miner or sandstone worker may contract silicosis in well under ten years and then very shortly afterwards tuberculosis may make its appearance. The coal miner has to wait for

twenty to twenty-five years for his lungs to show any ill effects from his work, and then he is more immune than the ordinary individual to infection by Koch's bacillus.

It is said that the silica particle damages the lung by chemical and not mechanical action, but theoretically the resultant trauma should easily be demonstrated.

The sharp-pointed silica molecule contained in such great percentage in the dust of gold mines has no soft, flocculent coal to surround the hard, dangerous edges, with the result that the bronchioles of the miner are torn and damaged even to a hæmorrhagic state and the nidus for tuberculosis growth is thus exposed and open. In the coal miner the linings have been coated with a protective material, with the result that, in his case, it is usual that fifty years of life have been completed before any disability is noticeable.

Prevention, of course, is the cure.

From records and statistics appended it seems that the only dangerous field in New South Wales and possibly in Australia is that lying along the hills of the South Coast of New South Wales.

The famous anthracite coal, which in pre-oil days fired the British Navy, is not in existence in Australia. The silica content of our soft coal is low. It varies only from 0·4% to 4·0%. It is really far inferior compared with that produced by the Welsh coal mines, and has much less irritating effect on the lung tissue. In the circumstances it is hardly right to compare the hazards of working in the Welsh coal mines with those of a mine of less than 1% silica.

Phobia.

There is one great thing in which we have to help the the coal miner. During the last few years there has grown up a terrific fear in coal miners working in the South Coast fields. The miners of the northern (Newcastle) and western (Lithgow) fields show less apprehension on this subject, and they have very little need to do so, for the industrial risk in those areas is possibly very comparable with that to men working in other so-called hazardous industries. The miners in the South Coast fields have lately contracted a neurosis, or hysterical outlook, which is quite unjustified.

The coal miner presents an economic problem. He belongs to a race whose ancestors have been born and brought up, lived and worked in the sooty atmosphere of mining towns and villages. His wife and children have been accustomed to dark cottages with a smoke-laden atmosphere and little sunlight, cobbled streets, wet days and the minimum amount of recreation. In Australia it is somewhat different, and the men who work in the coal mines in Ipswich, Queensland, glory in showing you their new rows of tidy, sunlit homes with gardens, well-paved streets, and within their homes modern conveniences. All this, however, does not get over the question that coal mining is a hazardous industry. From our statistics. it takes at least twenty—probably twenty-five—years for any man to contract fibrosis of the lungs, even in the worst mine in New South Wales. On the other hand, he may work in a pit all his life and show no sign of anthracosis. The miner sticks to his occupation apparently because it is an hereditary occupation. The quartz miner (gold, copper *et cetera*), if left to himself, can contract silicosis of his lung in three to seven years. He is a different individual; the adventure of prospecting does not enter into the coal miner's ambit.

At present in New South Wales there are three separate medical boards dealing with industrial hazards affecting the lungs. For many years the coal-mining industry was adjudicated upon by one set of medical men (the New South Wales Special Pneumonokoniosis Board), who had spent much of their spare time in intently studying this question of coal dust.

Unfortunately, some months ago, the South Coast miners were dissatisfied with the long-standing means of adjudication and asked for a separate group of medical men to be gathered together. The complaint was that the percentage assessment of disability had been under-estimated. Now there are two coal dust boards. The new board, dealing with the South Coast alone, has no radiologist attending its meetings, so that any discussion regarding the relation between the radiographic and clinical findings just does not occur.

The miners of the South Coast have lost interest in keen, critical discussion and in that scientific decision regarding their future that the old board was so anxious to give them.

The third medical board in New South Wales is one dealing with silicosis, meaning the lung infection found in men who have worked in gold, sandstone or any quartz, or in a similar dangerous occupation.

The important question of examining an applicant before employment and of deciding whether he should be allowed to work or not if he is suspected of pneumonokoniosis brings forward the right of the individual to decide his own future. If a "dusted" coal miner was a cause of infection to others, justification in preventing him from carrying on would be obvious. If, however, with the aid of his mate, the required number of daily "skips" are filled, and he is not spreading disease, can he be forced to relinquish his job?

Details of Examinations Made.

The following are the details of examinations made by the Special Pneumonokoniosis Board during the years.

Out of 316 miners sent for examination who worked exclusively in New South Wales coal mines, 223 were shown to have nodular fibrosis from a slight to a great degree. Of these 223, 94 were found to be totally incapacitated and 129 partially incapacitated. Of the 94, 77 were over the age of fifty years; thus 17 had become "dusted" before that age. Of the 129 who had some but not total incapacity, 87 had reached the age of fifty years; 42 were younger men.

Among those below the age of forty years, only two were found to be totally incapacitated and eight partially so. These ten all came from the southern field. There were no miners affected with "dust", either partially or totally, under the age of forty years in other areas.

Also out of the 223 who showed some signs, 129 came from the South Coast. The total number examined from this field amounted to 218, so this figure (129) represents 59% of the total examined.

In the northern coal field, out of 102 men examined, only 36 were found to have any sign of pneumonokoniosis (36%).

The western field produced 36 infected out of 58, which gives the extraordinarily high percentage of 62. In this district it was found that one mine in particular contributed to this high percentage.

The total number examined (58) in the western field is small compared with that (218) from the South Coast, and the members of the board have rather inclined to the idea that the South Coast is really somewhat more dangerous than the other fields. This has been substantiated by work carried out with the Division of Industrial Hygiene. A large number of miners have been clinically and radiologically examined, both in groups from one mine and in groups from different pits. As a result these investigations have shown that the incidence of anthracosis is somewhat greater in the South Coast than elsewhere.

Of men from other fields than the southern and of those who had worked in more than one field, 198 were examined; 47% had some dust invasion. This tends to demonstrate that the harder coal of the southern field is somewhat more deleterious than coal elsewhere. If the northern and western fields are compared with the southern and the statistics for "two or more fields" are disregarded, it is found that out of 160 miners examined in the former two fields 72, or 45%, were partially or totally incapacitated. This reduction of another 2% indicates that, in the mixed group, some of the men had worked in the southern and western fields.

Two hundred and seventy-six men examined had worked overseas as well as in New South Wales. Of this number,

164 were found to have contracted the disease to some extent, which makes the percentage of those affected 59. A small number (16) had worked in New South Wales and in other Australian States or in New Zealand; six of these were found to have some degree of pneumonokoniosis.

The following is a summary of the percentages of miners partially or totally incapacitated as a result of pneumonokoniosis:

(*a*) Southern field, New South Wales 59%
(*b*) Northern field, New South Wales .. 36%
(*c*) Western field, New South Wales 62%
(*d*) Two or more fields in New South Wales 58%
(*e*) Overseas and in New South Wales .. 53%

The average total number of employees (underground and surface) in New South Wales collieries during the five years ended June 30, 1946, was 17,600.

Of the miners found by the board in the same period to be suffering from pulmonary fibrosis due to dust, 242 were classed as partially incapacitated and 189 as totally incapacitated.

The total incidence of men with incapacitating pulmonary fibrosis due to dust was 4·90 per 1000 colliery employees per year. The incidence of those partially incapacitated was 2·75, and of those totally incapacitated 2·15 per 1000 colliery employees per year.

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There is hardly any necessity to mention the whole-hearted cooperation one has always received and the candid criticism that has been offered by one's fellow members of the Special Pneumonokoniosis Board. I am sure no team of workers could be more interested in all aspects of this subject.

I also have to thank His Honour Judge Perdriau for permission to use statistics obtained from the Workers' Compensation Commission.

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OCCLUSIVE ARTERIAL DISEASE.[1]

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IT is probable that vascular disease is as old as man. Undoubted evidence has been found in mummies of early Egyptian dynasties,(1) and the effects of occlusion were known to the Greeks in their descriptions of the senile gangrene of the aged.(2) The first accounts of morbid changes in blood vessels were made in the sixteenth century. Since then, although some knowledge has been gained, much remains unknown. To the pathologist, the cause and nature of early lesions are obscure; while the clinician can only hope to recognize the later stages and the effects of disturbed blood supply to tissues.

Disturbances of Blood Flow due to Physical Causes or to Disease.

Interference with the normal function of a blood vessel may arise in various ways: from an external cause such as injury, from distortion of the vessel itself by disease or from abnormal nervous influences. It is safe to assume that in almost every instance some damage to the vessel wall is an essential prelude to occlusion, the main exception being the lodging of an embolus.

Severance or contusion of an artery with resulting spasm will immediately place all tissues dependent upon the damaged vessel in grave danger. Unless an adequate collateral circulation is already established, a total death of tissue is inevitable. Appreciation of this fact determines the site for ligation in the arterial injuries sustained in battle.(3)(4) Other principles involved are the avoidance of ligation in continuity(5) and the desirability of tying the main vein if the principal artery of a limb is destroyed.(6)(7) In most instances the vein is injured at the same time, and if bruised it will thrombose later. The clot is likely to become infected and pyæmia may follow. Compression of an artery, if it occurs gradually as from the growth of a tumour, often permits the development of an adequate collateral circulation before complete occlusion occurs. In such circumstances the main channel may be obliterated without the production of any recognizable symptoms of altered circulation. Although the cause of much concern in times of war, trauma and compression of vessels are of relatively minor importance when compared with the disorders now to be considered.

Fallopius first described the degeneration of arteries into bone, and now the later stages of arterial disease can be differentiated and classified. It is customary to regard these changes as degenerative, but the evidence that blood vessels "wear out" is not convincing. Even the term "arteriosclerosis" is confusing, as it is often loosely used to include a variety of conditions which are capable of separation on histological appearances.

The most important both in frequency and in its effects is the disorder known as atheroma. The earliest signs of this process were described by Virchow(8) thus:

> There occurs a certain loosening of the connective tissue ground substance of the intima which must be attributed in a large measure to the increased imbibition of fluid elements from the passing blood stream.

Aschoff,(9) elaborating Virchow's theory, postulates two factors in the development of atheroma: first, an abnormal increase in imbibition and a certain slowing of the plasma which normally nourishes the inner layers of the vessel wall; second, a sufficient concentration of lipoids, especially cholesterol esters, in the plasma.

The cause of such changes is unknown, and from the wealth of investigation and experiment on the subject there emerge two principal theories, the mechanical and the metabolic. The mechanical theory is based upon the effects of the constant pulsating stress imposed on vessel walls throughout life. The preponderance of lesions in the lower extremities of man has experimental confirmation in the work of Klotz,(10) who produced intimal changes in the forelimbs of rabbits by suspending them by the hind legs for many days. Albutt sought to explain the site of intimal change on mechanical grounds by suggesting a shearing of the intima on the media.(11) Krafka considers herniation of the intima to be responsible.(12) The relation between hypertension and vascular change is confused and as yet has not been defined.

The metabolic theory is based upon the relationship between disorders of lipoid and cholesterol metabolism and vascular changes. Ample experimental evidence confirms the work of Saltykow,(13) Ignatowski(14) and Anitschkow,(15) who produced intimal lesions in animals, identical with those seen in man, by feeding them large amounts of cholesterol. However, too much reliance must not be placed upon the results of animal experiments, and it is wrong to draw exact analogies to the changes observed in man. Rabbits are naturally herbivorous and have difficulty in excreting cholesterol. Leary's(16)(17) observation on cholesterol-filled phagocytes in the lumen of the *vasa vasorum* of the human intima is important. He considers that these cells may be carried there by the blood from sites of injury, inflammation or metabolic overload. Efforts to correlate the incidence of human atherosclerosis with hypercholesterolæmia have not been successful. Bloor,(18) however, does not consider a high blood cholesterol level to be essential for the deposition of cholesterol in the arterial wall.

[1] A post-graduate lecture delivered on May 12, 1947, under the direction of the Melbourne Permanent Post-Graduate Committee.

Recently a third theory, which may be termed the vascular theory, has been advanced by Winternitz.[19] He has studied the blood supply of the vessel wall and suggests that in some instances intramural hæmorrhage may be the initial lesion in atheromatous change. He makes a plea for the reaction of the tissues of the vessel wall to be regarded as inflammatory rather than as degenerative. He summarizes his thesis thus:

> Surely an approach to the problem which is based on recognition of the artery as a vascular or potentially vascular organ, and therefore subject to the same pathological processes to which other tissues are subject, may prove more fruitful than one in which the lesions are regarded as primarily "degenerative" or as the inevitable concomitants of age.

This conception is of great importance and further work must bring interesting results. The growing knowledge of the specific reactions of tissues to particular chemical substances of the Duran-Reynals phenomenon and of Menkin's work on the fixation factor and on leucotaxine, when applied to the study of arterial disease, may revolutionize all present ideas on this subject.

Arteriosclerosis, or more correctly atherosclerosis, is a frequent and widespread disorder in which atheromatous intimal changes are found in association with lesions of the media, usually of an atrophic character.[20] The cause of this condition is obscure. Whether the medial change is a primary reaction to stress as suggested by Thoma, or whether it is secondary to intimal disease or a result of disturbed blood supply to the muscularis, is unknown.

Another type of change is observed in medium-sized muscular arteries. It is essentially a degeneration of the medial coat associated with calcification and is known as Mönckeberg's sclerosis.[21] Opinions differ as to its relationship to atheroma. Experimentally such lesions have been produced by Josue[22] and Mönckeberg[23] by the continued intravenous injection of adrenaline.

Another group of disorders which is capable of differentiation both on clinical and on histological evidence is exemplified by *thromboangiitis obliterans* and *polyarteritis nodosa*. Although these conditions are dissimilar in many ways they both present the picture of acute or subacute inflammation in and about blood vessels. *Thromboangiitis obliterans*[24] affects only the vessels of the extremities, arteries and veins being involved in a localized, non-suppurative inflammation which proceeds to organic occlusion of the vessel. The cause of the disease is unknown. The lesions are characteristically segmental, normal areas being found between diseased segments. The lesion in the acute phase is characterized by intimal proliferation and infiltration of all coats with small round cells. Thrombosis with subsequent organization occurs, and in the vessel walls there is much fibroblastic activity with occasional giant-cell formation.

Polyarteritis nodosa[25] is a relatively uncommon condition. Affecting the medium-sized arteries, it is not confined to the extremities and is often associated with systemic illness and pyrexia. It usually runs a fatal course and is seldom diagnosed during life. Rich[26][27] has recently advanced the theory that this condition is a manifestation of an allergic hypersensitivity. The histological picture is that of an acute inflammatory reaction in the vessel wall with necrotic changes in the media and infiltration of the vessel walls with polymorphonuclear leucocytes.

Other inflammatory reactions in the vessel wall are to be seen in the course of some systemic infections—for example, syphilis, typhoid fever and rheumatic fever. Apart from involvement of the aorta, the effects of syphilis are found in small vessels. The initial lesion is a periarteritis which progresses to occlusion through involvement of the entire vessel. Another interesting response to systemic disease is that seen in rheumatic fever.[28][29] The aorta is affected, but the main incidence is found in small arteries and arterioles. In these, the entire vessel may be involved and remarkable changes occur in the intima, which becomes blocked with fibrin and later is revascularized. In view of the modern conception of rheumatic fever it is interesting to note Boyd's[30] opinion on this subject. He states that "the peripheral lesions resemble those of *periarteritis nodosa* rather than those of syphilis".

Abnormalities of the Nervous Control of Blood Vessels.

So far, this discussion has considered disturbances of the distribution of blood to the tissues by physical means or by the processes of disease. Another type of disorder arises from abnormalities of the nervous control of blood vessels. In 1862 Maurice Raynaud described a "variety of dry gangrene affecting the extremities which is impossible to explain by vascular obliteration". This he considered to be due to a "vice in the innervation of the capillary vessels". Although it is possible he included in his thesis some examples of *thromboangiitis obliterans*, he was the first to suggest the possibility of interference with the peripheral circulation by nervous influences. In recent years Lewis[31] has suggested that the primary fault lies in an abnormal sensitivity of the digital arteries to direct stimuli, particularly low temperatures. Some patients, however, exhibit peripheral spasm under the influence of emotion or nervous tension. This suggests that there is, in some instances at least, a disturbance of central vasomotor function. The relief afforded by sympathectomy supports this view. It should be remembered that in 1901 Hutchinson[32] recognized that colour change in the extremities could arise from various causes, and suggested that the term "Raynaud's phenomenon" should be applied to these cases. A clear account of the present state of knowledge on this subject has been given recently by Allen, Barker and Hines.[33]

The opposite type of functional disorder, vasodilatation, has been studied by Lewis[34] and his colleagues. Although not especially relevant to the present subject, the effects of prolonged vasodilatation in producing vascular occlusion have been shown in the disease known as "immersion foot".[35] The peripheral vasodilatation which follows exposure to low temperature is associated with conglutination of corpuscles within the capillaries. Restoration of a normal circulation at a later stage is prevented, at times, by physical blockage of vessels with blood corpuscles. Another example of this type of obstruction occurs in cerebral malaria, but for a different reason: there is no antecedent vascular disease, but a "stickiness" develops in the red blood corpuscles which then agglutinate and effectively obstruct the circulation.

The Mechanism of Occlusion.

The main groups of conditions which may be associated with interference of the flow of blood to tissues having been reviewed, it is now necessary to consider more precisely the actual mechanism of occlusion. It is well to remember that man's survival of birth is dependent to a large extent upon the closure of the *ductus arteriosus*. The mechanism of this process is obscure, but the subsequent obliteration of the channel is associated with intimal proliferation and hyalinization identical with the changes described originally by Virchow.

The injection of large doses of thrombin into the circulating blood stream may cause extensive intravascular clotting; but if the thrombin is injected slowly it can cause complete defibrination without thrombosis. There is little evidence that normal blood contains any agent which inhibits the coagulation reaction.[36] Slowing of circulation, damage to the endothelial lining of a vessel or a combination of these two factors tends to produce the formation of clot. Platelets are deposited in roughened areas or at the sites of eddy formation. Local production of thromboplastins and of thrombin occurs, and a fibrin network forms which entangles red corpuscles. No doubt there are other factors yet unknown which may influence this process: the viscosity of blood, and the effects of exertion, of temperature and of nervous control, may all contribute to a slowing of the blood stream and to the production of conditions favourable for the deposition of clot. Observation of the extreme spasm seen on exposure of an injured artery leads to speculation as to the part

played by the nervous system in the production of coronary thrombosis. This supports the adage that "worry wears out the heart".

External pressure as by tumour growth, mechanical causes such as cervical rib or to a lesser extent the pressure of œdema fluid under tension in fascial planes, may contribute to slowing of the blood flow in the main vessels. If pressure develops gradually an expansion of collateral circulation occurs and an adequate circulation is maintained. If the pressure is severe and of sudden onset, gross damage to tissues may follow from circulatory impairment.

The sudden blockage of an artery by an embolus liberated from a distant source is often followed by changes similar to those observed under experimental conditions. The phenomena which occur after the complete arrest of the blood flow to a normal limb have been studied in detail by Lewis.[37] Immediately circulation ceases, the surface temperature begins to fall, the rate being determined by the difference between the temperature of the skin and that of the surrounding air. It may take many hours before thermal equilibrium is established. Colour changes are seen within a few minutes. A slight pallor develops at once and gives way to cyanosis within a variable length of time, depending upon the warmth, the elevation and the initial colour of the skin. This corresponds to the dilatation of minute vessels under the influence of metabolites. Almost simultaneously, small areas of blanching appear which slowly increase, producing a mottled appearance on the background of cyanosis. If the venous outlet from the limb is unimpaired, these white areas coalesce and eventually the skin becomes uniformly blanched.

Some evidence of interference with nervous function occurs within a few minutes of cessation of the blood flow. Numbness and tingling, which slowly spread centripetally, are followed by hypoæsthesia and anæsthesia. Paralysis of muscles occurs within a quarter to half an hour. Earlier, muscular contraction is possible; but if this is continued for a short period there develops a severe and constant pain from the accumulation of metabolites. This pain is frequently referred to as cramp, but careful observation will reveal the muscles to be in a flaccid state.[38]

If the period of ischæmia is short, between half an hour and one hour, restoration of circulation is followed by complete and rapid recovery of function. A transient reactive hyperæmia and some numbness and tingling are experienced during the recovery phase. When the period of ischæmia is prolonged, the nutrition of the tissues is permanently affected. Although nerves lose their function earlier than muscles, continued ischæmia kills muscle more rapidly than it kills nerve. Muscle fibres will die if deprived of blood for six to eight hours, and a replacement fibrosis follows which is recognized by clinicians as Volkmann's contracture. Nervous tissue can recover after a period of ischæmia of twelve to twenty hours. Examination of the skin will show weal formation after twelve hours, which leads to blistering and finally to necrosis if the circulation is interrupted for longer than twenty-four to thirty-six hours.

The tissue changes which occur as a result of occlusive vascular disease depend on several factors: the rate at which occlusion occurs, the presence of endarteritic changes in the vessels, and the degree of development and adequacy of the collateral channels which assume the responsibility for continuing the circulation. Although appreciable changes in the vessel walls can be produced in laboratory animals within a short space of time, it is generally thought that the process of human atherosclerosis takes years for its development to a stage in which gross interference with circulation is likely. Frequently the terminal event is intraarterial thrombosis, which arises either from clot deposition in a damaged and narrowed lumen or by a hæmorrhage in the vessel wall which may completely block the lumen.

The symptoms which develop will depend upon the nourishment of the tissues distal to the occlusion. Thrombosis of the middle cerebral artery or of one of its branches is followed by a dramatic loss of function, which is often permanent. Thrombosis of a branch of the coronary system may result in sudden death or in comparatively slight dysfunction, according to the size of the branch involved and to the degree to which anastomotic channels have developed prior to the occlusion.[39] Similarly thrombosis of the popliteal artery may in one instance cause massive tissue death with eventual loss of the leg, while in another it may pass almost unnoticed. The more gradual the development of intimal change, the greater is the possibility of an adequate anastomotic collateral circulation.

The intravascular thrombus undergoes organization, and in many instances this process is associated with the development of new blood vessels, which weave through the vessel wall and the clot in a haphazard fashion. To this process the term canalization has been applied. It is doubtful how much such channels contribute to the future blood supply of the distal tissues. Winternitz, by injection of the vessel walls, has shown their extraordinary ramifications in the region of organized thrombi. Ultimately the thrombosed vessel undergoes a hyaline and fibrotic type of change and shrivels into a thin, functionless cord.

It must be realized that on occasions vascular disease can proceed to total occlusion, such as has been described, without any previous warning of its existence. Usually, however, some suggestive signs or symptoms occur. Even so, there is as yet little that can be done to arrest the progress of vascular degeneration.

Signs and Symptoms of Occlusive Vascular Disease.

With the possible exception of localized pain or tenderness in the region of vessels subject to acute arteritis or phlebitis, no symptoms referable to the vessels are to be elicited at any stage of occlusive vascular disease. For years the morbid processes in intima and media proceed with no suggestive symptoms, and even the theory that the pain following embolus is due to vessel spasm is not yet firmly established.[40]

Accordingly vascular disease must be suspected and assessed almost entirely by symptoms and signs which arise in other tissues as a result of impairment of circulation. Disturbed nutrition of organs such as the brain and the heart will produce characteristic evidences of dysfunction. In the extremities, pain, colour changes, trophic disturbances and alterations of temperature and pulsation of vessels suggest disease of the peripheral circulation.

Investigation of the circulatory efficiency of viscera such as the brain and the heart is almost completely beyond the realm of clinical investigation. Abnormalities of motor, sensory or psychic behaviour may, on the basis of experience, suggest an impending cerebral catastrophe or the insidious onset of dementia. The occurrence of grave cerebral symptoms of transient duration, sometimes referred to as hypertensive crises, raises the question of the part played by vascular spasm in their production. Walshe,[41] on clinical grounds, considers that such spasm is likely, although this offers no explanation as to why it occurs only in the presence of vascular disease or of hypertension and not in normal vessels.

The occurrence of pain and dyspnœa related to exertion may lead to the suspicion of coronary sclerosis. Again the investigation of such a possibility is almost beyond present resources. Changes in the electrocardiogram can be produced by interference with the blood supply of the heart; but Meakins[42] issues a warning against too great a reliance on electrocardiography, thus:

> No matter how meticulously the *QRS* and *T* deflections are studied, the knowledge so obtained is but limited, and what is worse it becomes almost Delphic in its oracular dogmatism, platitude being piled upon platitude.

The relief afforded in some cases of *angina pectoris* by cervical sympathectomy suggests that, as in the brain, spasm of diseased vessels may at times contribute to the production of symptoms.

Circulatory disturbances of the extremities allow of more exact investigation by instrumental means, and much of our knowledge of these disorders has come from observa-

tions of Lewis and others[43][44] on the peripheral vessels. The pain which arises in a poorly nourished limb is of two types: one which occurs at rest and which is not exaggerated by moderate exercise; the other, often referred to as intermittent claudication, bearing a definite relation to muscular exertion. The cause of the former pain is obscure, but it is thought to be due to cross-stimulation of sympathetic and sensory fibres or to increasing digital thrombosis. It is often dramatically relieved by sympathectomy. The pain of claudication, which at one time was thought to be due to vascular spasm, is not relieved by interference with the sympathetic nerve supply, and has been shown by Lewis to be due to the accumulation of a metabolic product, factor *P*, which stimulates sensory nerve endings in muscle.

The colour changes consequent upon disturbances of circulation have been clearly described and explained by Lewis. Appreciation of the significance of pallor, rubor and cyanosis when considered with the position and temperature of the limb yields valuable information as to the state of circulatory activity.

Not only is the temperature of the skin of an extremity a guide to the circulation therein, but it can be made a useful agent for the study of the patency of the main vessels. The observation of reflex vasodilatation of the skin of an extremity following heating of the trunk, nerve block and spinal anæsthesia is a sound method of distinguishing between organic vascular disease and certain vasospastic states.

Examination of the vessels themselves provides further information. The presence of pulsation in the main arteries, the rate of filling of veins after elevation of the limb, and the effects of reflex hyperæmia after occlusion of the main vessel when the limb is warmed, all provide additional evidence. More exact instrumental means of determining the blood supply are provided by the use of an oscillometer or a plethysmograph. X-ray examination may give evidence of disease by revealing the calcified outline of diseased vessels. Intraarterial injection of a contrast medium such as "Per-Abrodil" permits the outlining of the vascular tree, and in skilled hands provides valuable information. This procedure is not without some element of risk, and gangrene of an extremity has occurred as the result of subsequent intraarterial thrombosis.

The general nutrition and muscular development of the extremity must not be disregarded. Trophic changes in the nails and in the skin of the digits are of importance. The skin of an extremity which is poorly nourished is easily injured. Elaboration of H substance at the site of trauma, instead of being of benefit, may further slow a flagging circulation and thus precipitate tissue necrosis or gangrene.

Treatment.

Until the cause of atherosclerosis is known there can be no reasoned treatment of vascular disease. By the time vascular changes are suspected or recognized, the morbid processes are well advanced, and little can be done other than an attempt to delay or relieve the effects of impaired circulation. Much speculation has been made concerning the relationship of *diabetes mellitus* to the development of atherosclerotic changes. There is no doubt that with the increased expectation of life which has resulted from modern treatment the diabetic has been reprieved, only to develop vascular disease at an earlier age and in much greater proportion than other members of the community. There is a growing school of thought which maintains that strict control of the diabetic state obviates or delays the onset of these vascular changes. This contention represents a possible influence on one cause of arterial disease.

In those disorders in which disturbances of nervous control occur, interference with the sympathetic nerve supply will result in cutaneous vasodilatation, and in appropriate cases symptomatic relief or cure will follow. It must be realized that paraganglionectomy is not a cure in peripheral vascular disease. Its limitations and the indications for its performance have been summarized recently by Telford.[45] There is no evidence to suggest that sympathectomy has any effect upon the processes of the disease in the vessels. It is also necessary to remember that the main benefit of sympathectomy is found in the cutaneous circulation, and that relatively little alteration in the blood supply to the muscles or deeper structures follows such an operation.

In recent years it has been found possible to influence the clotting time of blood by the use of a phosphatide isolated from the liver. This substance, known as heparin, acts by preventing the activation of prothrombin to thrombin and also by neutralizing the action of thrombin itself. A second substance isolated from clover and named "Dicumarol" possesses the property of reducing the plasma prothrombin concentration. With the development of a simple method of determining the blood prothrombin level, it is now possible to use both heparin and "Dicumarol" in order to alter the coagulation time of the blood. The place of these substances in the treatment of thrombotic accidents has yet to be established; but reports so far appear to indicate that there is a possibility of influencing thrombosis by the properly controlled use of these drugs.

An empirical procedure which may have its justification in influencing the viscosity of the blood is the frequent intravenous injection of small quantities of hypertonic saline solution. Opinions are divided as to its efficacy and as to its mode of action. In some instances its use has proved of undoubted benefit.

A large number of drugs and tissue extracts have been examined for possible action upon the blood vessels or the nervous control of the circulation. These are reviewed in a monograph by Abramson.[46] It can be said that at present there is no outstanding benefit to be obtained from any known preparation.

Apart from the general measures which have been mentioned, numerous procedures are employed in the treatment of peripheral vascular disease to stimulate the circulation in the extremities. Graduated exercises, oscillating beds, intermittent vascular occlusion, reflex vasodilatation and others all find legitimate use. Detailed descriptions are beyond the scope of this discussion.

Conclusion.

In its present state of development, medicine is still more concerned with the healing of disease than with its prevention. So with vascular disease, we realize that our attempts at treatment are limited to influencing and delaying the effects of processes beyond our knowledge and control. A greater understanding of the phenomena of inflammation, of the influences of heredity and of the effects of nutrition will enable us, in time, to control and possibly reverse the morbid changes here described.

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ALCAPTONURIA, WITH A REPORT OF AN ADDITIONAL CASE.

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Alcaptonuria is a condition of disordered metabolism, characterized by the presence of homogentisic acid, (dihydroxyphenyl acetic acid) in the urine. It is a very rare condition, and up to the present time, 146 cases have been recorded (Cockayne[1]) in the literature, chiefly from Germany, England and America. It occurs more frequently in males than in females, in the proportion of 100 to 46.

The present case appears to be the first of its kind to have been reported in Australia, and brings the total in the world's literature to 147.

Alcaptonuria is a disorder of catabolism of the aromatic amino-acids, tyrosine and phenylalanine, as the enzyme system required to break down the benzene ring is missing and utilization of these amino-acids stops at this stage. It has been called by Garrod[2] an "inborn error of metabolism", and classed with cystinuria and albinism.

Alcaptonuria is characterized by the continued presence, throughout the life of the individual, of homogentisic acid in the urine, and depends upon a single gene defect. This may arise *de novo* as a mutation, or it may be inherited as a Mendelian recessive character.

It is, in fact, of great scientific interest, and is the best-known example of the inheritance of a metabolic defect according to the Mendelian laws. In common with other Mendelian recessives, it is found more frequently in males than in females, and in the offspring of consanguineous marriages. Since the gene defect has to be present in both gametes to produce this disorder, it will be seen that the union of first cousins will provide the greatest likelihood of these circumstances.

The only exception to this rule is the family investigated by Pieter,[3] in which the condition appeared to be inherited as a dominant.

Alcaptonuria may be noticed in early life by the urine staining the diaper a brown or black colour; but many subjects reach adult life before the condition is detected. As the alcaptonuric reaches middle age, the fibrous tissue, cartilage and synovial membranes may be stained a black colour, and the sclerotics may show a brownish pigmentation. This was first described by Virchow in 1841, a condition known as ochronosis. A form of osteoarthritis involving especially the spine and hip joints may occur, and leads to a peculiar waddling gait, as in the case of two brothers described by Osler[4] in 1904. It will thus be seen that a minor defect of metabolism, after many years, may lead to a permanent disability.

It was assumed by earlier writers that alcaptonuria also caused arteriosclerosis; but as the latter is common after middle age, the incidence among alcaptonurics does not appear to be higher than among other individuals of the same age group.

The urine of an alcaptonuric may appear of normal colour on being passed, but turns pink, brown and finally black in layers from the surface downwards, owing to oxidation. This colour change is prevented by excluding the contact of air by adding a layer of toluol, and is hastened by heating and especially by the addition of a caustic alkali—for example, a few drops of sodium hydroxide solution.

The urine will reduce Fehling's and Benedict's solutions, the appearance of a yellow deposit against a black background being characteristic. It will reduce an ammoniacal solution of silver nitrate even in the cold; but it does not cause rotation of polarized light, nor does it ferment with yeast, since the reducing substance is not a sugar but an aromatic acid.

A characteristic reaction is given on the addition, drop by drop, of very dilute ferric chloride solution, a transient blue or greenish colour appearing. This colour reaction will not appear if the ferric chloride solution is too strong, as the oxidation takes place too rapidly.

Alcaptonuria has been detected in other vertebrates besides man—for example, in cattle (Fink),[5] in a rabbit (Lewis)[6] and in a sheep (Priestley).[7]

The clinical significance of this condition is chiefly its differentiation from *diabetes mellitus*, the mistake having been made many times in the past. The reactions of the urine and a normal glucose tolerance curve will differentiate between the two conditions.

Alcaptonuria is of great importance in human genetics, and most detailed studies of cases have been made, for example by Hogben *et alii*.[8]

Report of a Case.

Mrs. I.A., a widow, was born in Glasgow fifty-six years ago, her parents being first cousins. She has three brothers and one sister living in Scotland, and they appear to be unaffected. Her own marriage was childless. She had noticed that since childhood her urine had stained her clothing a brownish colour. Two years ago she consulted her medical attendant on account of backache in the lumbar region.

On examination of the patient, the cartilages of her ears and nose appeared to be a bluish-black colour, and bilateral symmetrical brownish areas, five millimetres in diameter, were seen on the sclerotics lateral to the corneo-scleral junction. There was some degree of rigidity of the spine, but the gait was unaffected. The blood pressure readings were 150 millimetres of mercury, systolic, and 80 millimetres of mercury, diastolic. The urine was a pink colour and turned black from the surface downwards on exposure to the air. The urine reduced Benedict's solution and gave the characteristic colour change with dilute ferric chloride solution. An X-ray examination showed evidence of osteo-arthritis of the lumbar vertebræ. The glucose tolerance curve was normal.

Summary.

1. The condition of altered metabolism known as alcaptonuria has been briefly discussed.

2. Its clinical and genetic importance has been indicated.

3. An additional case, the first of its kind in Australia, has been reported.

Acknowledgements.

I wish to thank Professor H. Priestley and Dr. H. S. H. Wardlaw for their advice in the preparation of this paper.

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A METHOD FOR THE EQUILIBRATION OF BLOOD WITH A GAS MIXTURE.

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IN the course of a recent investigation, it was necessary to produce a series of carbon dioxide dissociation curves for human blood. The laboratory procedures involved in the construction of such a curve are: (i) equilibration of the blood with a gas mixture of known composition; (ii) analysis of the blood for its corresponding carbon dioxide content.

One of the earliest comprehensive papers on this subject was published by Christiansen, Douglas and Haldane[1] in 1914. For the equilibration of the blood with gas mixtures, these workers used an apparatus which consisted of a cylindrical saturating flask of 400 millilitres capacity containing the blood specimen and the gas mixture. This was placed in a water bath at 37° C., and rotated for five minutes by means of a motor. The pressure within the flask was then adjusted to atmospheric level and rotation was continued for a further ten minutes. In order to obtain samples of blood and gas for analysis, the flask was removed from the water bath and wrapped in a warm cloth. A sample of the gas was then taken into the ten millimetre burette of a Haldane gas analysis apparatus, and a sample of the blood was sucked into a one millilitre graduated pipette. The analysis of the blood was carried out according to the method of Barcroft and Haldane.[2]

In this department the method used for blood analysis is that described by Van Slyke and Neill.[6]

As for the equilibration of the blood with the gas mixture, for some time the method used was that described above, but certain technical difficulties made necessary its modification. Stadie[7] had also found modification necessary.

The difficulties which we met were: (i) the inevitable fall of temperature in the saturating flask after its removal from the water bath with consequent reduction in pressure; (ii) the further fall in pressure occasioned by the withdrawal of the gas sample; (iii) the frequency with which contamination with extraneous air bubbles occurred during transference of the blood from saturator to pipette. This introduced two sources of error complementary to one another: (*a*) alteration of the gaseous tension of the blood by contact with the air and air bubbles, and (*b*) inaccuracy in the volume of the blood sample in the pipette due to the presence of minute air bubbles swept in from the connexions.

Apparatus.

In an effort to overcome these technical difficulties, an apparatus was designed which took definitive form as follows (Figure I).

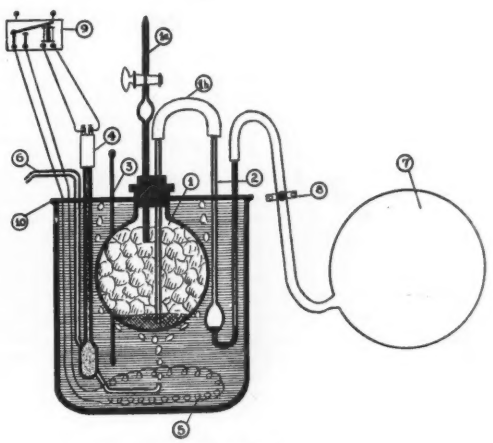

FIGURE I.

1: Bolt-head flask; 1a: blood pipette; 1b: capillary tube; 2: U-tube; 3: thermometer; 4: benzene-mercury thermostat; 5: heating element; 6: air bubbler; 7: football bladder; 8: tap; 9: electro-magnetically operated mercury relay switch; 10: lid.

A cylindrical glass container approximately eight inches in length and five inches in diameter, surmounted by a wooden lid, was used as a water bath. Supported by the lid and immersed in the water were:

1. A short-necked bolt-head flask of 400 millilitres capacity. In the tight-fitting rubber stopper in this flask two holes were pierced. Through one of these an inverted blood pipette was thrust for a distance of one inch. The other carried a finely drawn out capillary tube, one end of which extended downwards to within one millimetre of the bottom of the flask. The other end was connected by rubber tubing with item 2.

2. A U-tube, one arm of which was expanded into a bulb. Several millilitres of distilled water were placed in the U-tube. Connected by rubber tubing with the arm of the U-tube remote from the bulb was a football bladder containing the desired gas mixture.

3. A centigrade thermometer.

4. A mercury-benzene thermostat (Thompson[8]) which by making and breaking a four volt direct current circuit activated the electromagnets operating a mercury relay switch. This switch controlled the supply of current (40 volt alternating current) to item 5.

5. A jug heating element placed near the bottom of the water bath. By means of the thermostat the temperature of the water was maintained at 37° ± 0·1° C.

6. A length of glass tubing by means of which air was bubbled through the water. This ensured mixing and the maintenance of a constant temperature throughout the water bath.

Procedure.

Collection of Blood.

Twenty millilitres of blood were drawn off without stasis from the patient's antecubital vein into a syringe lubricated with paraffin and containing a small amount of heparin. At first heparin was the only anticoagulant used, but it was found that, after standing for a period, this heparinized blood contained minute masses of fibrin. To prevent this, an additional anticoagulant—neutral potassium oxalate—was used as follows. Three millilitres of a solution containing 1·843 grammes *per centum* of neutral potassium oxalate and 0·84 gramme *per centum* of neutral sodium fluoride (Harrison[5]) were placed in a small bottle and deposited by evaporation on its sides. The presence of the sodium fluoride in the solution inhibited glycolysis (Lovatt Evans[6]). Previously, a small amount of crystalline sodium fluoride had been placed in the syringe with the heparin for this purpose. The blood was transferred from the syringe to the bottle and shaken.

Equilibration of Blood.

With the apparatus set up as described above, both electrical circuits were switched on and the air-bubbler was turned on. When the temperature of the water bath had reached 37° C. the blood was placed in the bolt-head flask.

Three to four litres of a mixture of carbon dioxide and oxygen in the desired proportions were introduced into the football bladder from a cylinder. For convenience we had gas mixture supplied in bulk in 100 cubic feet cylinders. One of these contained approximately 5% carbon dioxide in pure oxygen, and the other approximately 10% carbon dioxide in pure oxygen.

The bladder was connected with the U-tube and the intervening tap gently turned on. The rate of flow of gas was such that the bubbles passing through the water in the bulb of the U-tube could just be counted. The passage of the gas through this water also served to saturate it with water vapour at 37° C.

The gas, on issuing from the capillary near the bottom of the bolt-head flask, passed upwards through the blood and caused it to froth. This froth displaced the air from the flask upwards through the blood pipette, the tap of which had been left open. The frothing of the blood was also a means of presenting a large surface area to the equilibrating gases, since, at the instant just before the individual bubbles collapse, the blood film forming its wall can hardly be thicker than the dimensions of one corpuscle. The rate of influx of the equilibrating gas was adjusted so as to maintain the froth level at a point just below the lower end of the blood pipette. It was found that equilibrium between blood and gas was not attained until approximately two litres of the gas mixture had been passed through the blood. This took at least fifteen minutes. The influx of the gas was then temporarily stopped until the froth had almost subsided. Just before withdrawal of the blood sample, a small volume of gas was vigorously bubbled through the blood to counteract any tendency to sedimentation. The gas supply was then turned off.

Withdrawal of Samples.

Blood.—A dry pipette, previously warmed to 37° C. in an incubator, was substituted for the one already in position. It was thrust downwards until the end came to rest below the surface of the blood, almost touching the bottom of the flask. A sample of blood sufficient almost to fill the pipette was then drawn off by oral suction and the pipette tap closed. The pipette was then withdrawn through the stopper and its contents were adjusted to the required volume by running off the excess blood. The sample was then transferred as quickly as possible to the extraction chamber of the Van Slyke manometric apparatus.

Gas.—The gas sample for exact analysis was withdrawn directly from the football bladder into an evacuated gas sampler and thence into a Haldane gas analysis apparatus (Haldane and Graham[4]); for it had previously been found by a series of analyses that after approximately two litres of the equilibrating gas mixture had been passed through the blood there was no detectable difference between the composition of the gas drawn directly from the football bladder and that of the gas taken from the flask.

Discussion.

With this method of equilibration, there is no possibility of a change in either the temperature or the pressure in the flask system during the withdrawal of the blood and gas samples. At no stage during the equilibration does the pressure of the gases above the blood within the bolt-head flask vary from atmospheric level. The total pressure within each individual bubble, however, does exceed atmospheric pressure by a quantity expressed in the formula $p = \frac{4S}{r}$ where p is the excess of pressure, S the surface tension in the wall of the bubble, and r the radius of the bubble (Booth and Nicol[2]). This, however, is a quantity so very small as to be of no present significance.

Frothing is known to produce concentration and, in some circumstances, coagulation of protein at the liquid-gas interface. The possible effect of this on the gaseous content of the blood was tested by experiment over a period of two and a half hours with an entirely negative result.

In the Haldane equilibrating apparatus, there was always the possibility, if not the certainty, of the occurrence of a fall of temperature in the saturator flask after its withdrawal from the water bath. This source of error is obviated in the method described above.

The changing of the blood pipettes just prior to withdrawal of the blood sample prevents any dilution of the blood with water vapour which may have condensed on the walls of the pipette during the time of equilibration. The complete saturation with water vapour of the gas mixture entering the blood offsets any concentrating effect which loss of water vapour in the gas leaving the blood might have. In any case the volume entailed is very small.

The final source of difficulty mentioned as being encountered in the Haldane method was the occurrence of air bubbles and the contamination of the blood with air in the connexions. In the apparatus herein described the pipette is thrust directly into the blood, the necessity for any intermediate connexion being thus eliminated. A further safeguard against contamination of the blood is the fact that, since there is a constant stream outwards of the equilibrating gas via the pipette, it is itself filled with the equilibrating gas and not with air. Thus, in the withdrawal of the blood sample, the only gas which it encounters is that with which it is already in equilibrium.

It is mainly for the equilibration of blood with mixtures of carbon dioxide and oxygen that we have used this method, but any mixture of gases can be used.

Summary.

1. A method is described for the equilibration of blood with a gas mixture.

2. An attempt has been made to avoid certain difficulties inherent in other methods.

3. The possibility of a fall in temperature during the transfer of blood from the equilibrator to the pipette has been eliminated. Likewise, throughout the entire procedure, the pressure within the equilibrator remains constant.

4. Contamination of the blood sample with extraneous air is avoided.

Acknowledgements.

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Reports of Cases.

TWO CASES OF EOSINOPHILIC GRANULOMA OF THE LOWER EXTREMITY RESEMBLING PARAMYCETOMA.

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DURING the year 1947 two full-blooded aboriginal male infants, aged approximately two years, were admitted to the Alice Springs Hospital with diffuse enlargement of the lower extremity. The lesions were similar both in location and in clinical appearance, Later, the histological pictures of sections prepared from both patients also proved the same. It was merely a coincidence that these two patients were admitted to hospital at the same time, as they were children who came from two different isolated cattle stations over 500 miles apart.

From the case reports to follow it will be realized that the diagnosis of the disease from the clinical appearances alone could only be speculative. Obviously special investigations through the laboratory would be necessary to make and confirm the diagnosis.

Case I.

A male aboriginal, aged about two years, was admitted to the Alice Springs Hospital on February 25, 1947. He came from Newry Station, situated 329° north-west of and 600 miles from Alice Springs, at 16° 1′ 26″ south latitude and 129° 15′ 0″ east longitude. On inspection of the patient the left thigh and buttock were seen to be symmetrically enlarged. On palpation the swelling was diffuse and involved the tissues under the skin. The infiltration was hard, rubbery and painless. In some areas it formed nodules and plaques, while in other places a more diffuse infiltration occurred as a result of coalescence. These lesions extended beyond the left iliac crest to the lower flank.

The general appearance was good. The temperature and pulse rate were normal. There was no enlargement of the lymph glands, spleen or liver. Examination of the child's blood gave the following information. The hæmoglobin value was 70% (Sahli). The red blood cells numbered 4,500,000 per cubic millimetre. The white blood cells numbered 30,000 per cubic millimetre; 0·5% were juvenile cells, 0·5% band cells, 46·5% neutrophile cells, 34% lymphocytes, 17·5% eosinophile cells and 1% mononuclear cells. No parasites were seen in the blood films. Stool examinations revealed the presence of Strongyloides larvæ.

Specimens of tissue taken at varying levels were reported on as follows by Dr. T. C. Backhouse, of the School of Public Health and Tropical Medicine, Sydney:

> The epithelium and immediately subjacent corium show relatively little change. Deeper and extending to the subcutaneous fatty tissue is a dense infiltration of granulomatous character. There are small areas of necrosis with polymorphonuclear leucocytes predominating, areas of plasma cells and fibroblasts with scattered giant cells of foreign body type and throughout most areas numerous eosinophile leucocytes. No parasites or remains of these could be found in a large number of sections studied and no fungi or bacteria could be demonstrated in Gram or Ziehl-Neelsen preparations. The latter showed some amorphous acid-fast material.
>
> In general the tissue reaction resembles that found in the section from the boy "Johnnie" (Case II), although the eosinophile reaction is less intense.

Further biopsy sections were examined by Professor J. B. Cleland, of Adelaide, who stated in a personal communication that streptothrix elements were detected in the sections. A cultural examination of discharge collected from a broken-down nodule was attempted by Mr. E. French, of Adelaide, with negative results. Massive doses of potassium iodide were given, with considerable reduction of the lesion.

FIGURE I.
Map showing location of Newry and Elkedra Stations.

Case II.

An aboriginal male infant, aged two years, was admitted to hospital on January 26, 1947, from Elkedra Station, situated 30° north-east and 204 miles from Alice Springs, at 21° 7′ 3″ south latitude and 135° 25′ 0″ east longitude. The child's right thigh was swollen from the groin to the knee by a hard, rubbery and painless infiltration of the subcutaneous tissues. Within the diffuse swelling were a few nodules and plaques coalescent with the main lesion. No lymph gland, splenic or liver enlargement was detectable, and no deterioration had occurred in the patient's general condition. The temperature was normal.

Examination of the child's blood gave the following information. The hæmoglobin value was 70% (Sahli). The red blood cells numbered 4,000,000 per cubic millimetre. The white blood cells numbered 22,000 per cubic millimetre; 1% were band cells, 37% neutrophile cells, 48% lymphocytes, 10% eosinophile cells and 4% mononuclear cells. No parasites were seen in the blood films. Stool examinations revealed no larvæ or ova.

Specimens of tissue taken at different levels were reported on by Dr. A. H. Tebbutt, of Sydney, as follows:

> I find plasma cells numerous in places. The giant cells are of foreign body type and grouped in foci which must, I think, be related to some foreign particulate substance. This substance also determines a plasma cell and eosinophile cell infiltration at a distance, possibly by diffusion of the foreign substance, the eosinophile cell exudate being as it were an allergic response. I cannot see any definable foreign substance except a curious dirty reddish or pinkish substance in the hyaline cytoplasm of one or two giant cells. I see quite a few foreign body lesions; usually, but not always, there is some trauma or fat necrosis or possible entry of foreign bodies through the skin. In this case such a widespread lesion would be more in keeping with some living parasite, now possibly dead.

In regard to the later sections, Professor J. B. Cleland stated in a personal communication that the histological appearances were consistent with the infection's being due to a streptothrix, but no streptothrix threads were seen in the tissue.

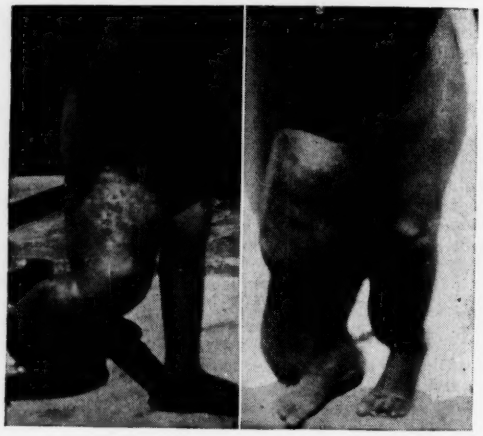

FIGURE II.
Photographs illustrating the lesion.

Sections of sterile tissues taken at the time of biopsy were ground in nutrient broth and incubated, with negative results.

The child was given massive doses of potassium iodide, with considerable diminution of the lesion.

Conclusions.

The lesions described would appear to be related to the mycetomatous group, in which the microscopic picture of the tumour masses corresponds to those of other similarly grouped infectious granulomata—namely, the formation of granulation and fibrous tissue with epithelioid and giant cells, round cells, cellular debris and frequently eosinophile and polymorphonuclear cells.

Castellani and Chalmers split off another group, the paramycetomata. These are characterized by the absence of granules in the discharge or affected tissues and a histological picture, in which the following features are present: (i) Eosinophilic bodies occur either enclosed in cells or lying free. (ii) Fungous elements are found, frequently nocardial hyphæ with or without the presence of spores, and fungi of the leptothrix type. (iii) Occasionally minute grains are observed, few in number. Most frequently grains are absent because the condition is caused by hyphal elements not collected compactly. (iv) Plasma cell infiltration is present.

Summary.

1. Two cases of elephantiasis of the lower extremity in aboriginal children are described.

The gross appearance of the abnormal tissue when cut was identical in both cases. The subcutaneous tissues were replaced by a dull, yellow-white substance, which was hard and rubbery to the cut. It resembled in appearance and consistency the common fibromyoma.

2. The histological pictures of the lesions were those of an eosinophilic granuloma.

3. The lesions were clinically and histologically related to the paramycetomatous group of fungous diseases.

4. The lesions responded well to massive doses of potassium iodide.

Acknowledgements.

I am indebted to Professor J. B. Cleland, Dr. T. C. Backhouse and Dr. A. H. Tebbutt for the determination of the infection and the histopathological evidence. My thanks are due to the Director-General of Health, Commonwealth Department of Health, Canberra, for permission to publish this paper.

CONGENITAL PREAURICULAR SINUSES: THREE CASES.

By J. M. O'DONNELL,
Perth.

Case I.

MRS. J., aged thirty years, had for about eight or nine years been troubled with an "abscess" in front of the left ear. It used to swell up and discharge, leaving behind a red raised scar. About six months prior to examination she had noticed a discharging "pore" on the front end of the rim of the ear (A in Figure I); from this "pore"

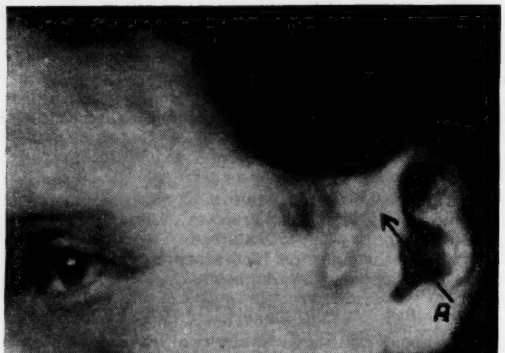

FIGURE I.

a discharge had been coming away which was at times offensive. In the last few weeks after a period of some months of inactivity, the scar had swelled up to form an abscess which had to be opened. The spot settled down again. Her general health is excellent.

Comment.

The lesion had been suspected of being tuberculous; this diagnosis had never been proved from examination of the pus, Mantoux test, *et cetera*. The response to the Wassermann test was negative. The lesion had received

various treatments and had had several courses of X-ray therapy with beneficial results. On several occasions intravenous injections of gold had appeared to be of benefit and to prolong the period of quiescence. The more or less recent manifestation of the sinus at the anterior end of the rim of the ear gave the clue to the diagnosis. The inconspicuous "pore" had not been noticed before.

There is no family history of similar trouble with the side of the face.

On the right ear there is also a vestigial remnant which takes the form of a pore or dimple; but it is not associated with any preauricular inflammatory condition.

Case II.

Miss McK., aged sixteen years, when seven years old developed a lump in front of the left ear. This was opened but did not heal. It was subsequently treated with ointments at the Children's Hospital, Perth, and eventually was reduced to a dry scar. When she was fifteen years old (December 12 prior to examination) the lump came up again and started to discharge; she was given penicillin ointment at the Royal Perth Hospital about a month before examination. She has also had a "discharging pore" (A in Figure II) on the rim of the

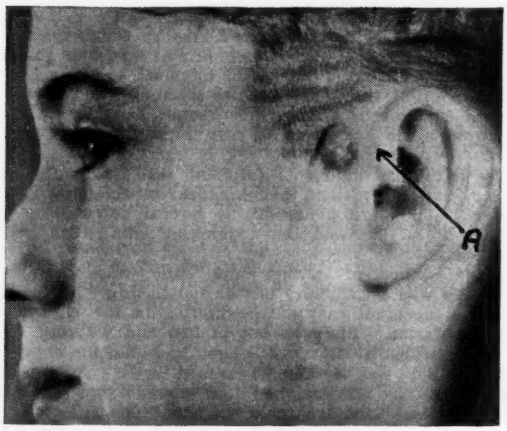

FIGURE II.

ear for as long as she can remember. The lump originally came up after she had received a kick on the spot while playing with another child. Her general health is good. She has few colds, but no bronchial colds and no sores elsewhere. Her weight is satisfactory.

Examination showed the patient to have a granuloma in front of the left ear and a vestigial dimple on the rim of the ear. This had a sinus which contained pus or sebaceous material. The granuloma was freely mobile on the underlying tissues and did not feel as if "tethered" to the anterior part of the ear by subcutaneous inflammatory processes. The two lesions appeared to be separate. No others in the family were known to have similar trouble. There was also a vestigial remnant in the form of a pore or dimple at the anterior end of the helix on the right side.

This patient has been since operated on by Dr. H. J. Gray. The vestigial pore, the track and the granuloma were excised. When she was last seen by me the result was excellent.

Case III.

Master R.G., aged nine and a half years, had had a sore on the left side of his face for two years. It came up and then discharged, after which it subsided for some months. His mother said that he had always had a "blackhead pore" on the front end of the rim of the ear. This discharged "blackhead type of pus" when squeezed. A piece of the sore had been cut out eight months earlier. The pore was also removed. Examination of the tissue revealed "tuberculoid tissue". A Mantoux test performed recently produced a strongly positive reaction with the first strength. Apart from the "sore" on his face, the child's general health and development were excellent.

No photograph of this patient is available.

No vestigial remnant was present on the right side.

Comment.

The sinuses are thought to be caused by incomplete fusion of the three nodules from which the tragus and helix are developed. These nodules are derived from the first branchial arch; there is a familial tendency and the lesions are congenital. In my cases the lesions were all on the left side.

Treatment is surgical. According to Weaver[1] a successful method is to inject the sinus with methylene blue and then dissect it out and coagulate the areas stained. Total extirpation of the whole tract is also practised when possible.

Conclusion.

The cases are reported because I believe them to be rare; these are the only cases I have encountered. I am also reporting them in the hope of receiving helpful suggestions on treatment.

Acknowledgements.

I wish to thank Dr. Harry Lucraft for the photographs, Dr. H. J. Gray for drawing my attention to the vestigial "pore" remnant in Case II, and Dr. R. S. W. Thomas for looking up the cases reported in *The Laryngoscope*.

Reference.

[1] D. F. Weaver: "Congenital Pre-auricular Sinuses", *The Laryngoscope*, May, 1946, page 246.

SECONDARY ABDOMINAL PREGNANCY WITH SURVIVAL OF THE FŒTUS AND MOTHER.

By G. H. MOORE,
Townsville, Queensland.

EXTRAUTERINE PREGNANCY, with its usual sequelæ of tubal abortion, tubal rupture or pelvic hæmatocele, is a fairly common condition; but continuation of the pregnancy to a late stage of fœtal development is very uncommon. The following case history describes such a condition with survival of the child and its mother.

Clinical Record.

Mrs. M.C.F., aged seventeen years, was admitted to the maternity ward of Townsville Hospital at 1 a.m. on February 20, 1947, this being her first attendance at the hospital. She said that she was pregnant, her last menstrual period having occurred in June, 1946. During the evening of February 19 she had had a feeling that her bowels wanted to act a lot, but they did not function very well. She then developed severe pain in the lower part of the abdomen spreading quickly all over the abdomen, and thought she was in labour. There had been no hæmorrhage or discharge of fluid. The medical officer on duty examined her and was satisfied that she was not in labour. He noted that the abdomen was difficult to palpate on account of tenderness and muscular guarding. The uterus appeared to be of about thirty-four weeks' gestation; the fœtus was palpated, and was thought to be presenting by the breech and lying on the right side of the uterus. The patient's temperature was 99° F. and her pulse rate was 100 per minute.

At nine o'clock on the morning of February 20 I saw the patient and examined her. She was a well-developed, muscular girl, rather excitable at the time and complaining

of slight generalized abdominal pain. The temperature was 99° F. and the pulse rate 120 per minute. Her colour was normal and she did not appear ill. The breasts were large and contained colostrum. The heart and lungs were normal on clinical examination. The blood pressure was 140 millimetres of mercury, systolic, and 80 millimetres, diastolic.

The abdomen was enlarged by a swelling the size of a thirty-four weeks' pregnancy, but difficult to examine on account of guarding. There seemed to be diffuse tenderness but no rigidity. The outline of the uterus could not be defined properly, and no uterine contractions were felt; but the fœtus was palpated lying on the right side of the uterus and presenting by the breech. The breech had not engaged in the pelvis. The fœtal heart sounds were normal. There was no vaginal discharge or "show". The urine was normal to ordinary ward tests. An enema was ordered, a good fæcal result was obtained and the patient felt much better.

At six o'clock on the same evening the temperature was still 99° F., but the pulse rate had increased to 136 per minute. Pain in the abdomen was much less severe. Slight tenderness of the whole abdomen was still present, and guarding was present on palpation. No uterine contractions were felt. The fœtus was in the same position. On February 21 the patient felt well and was allowed out of bed. She had no pain. The fœtus was still in the right sacro-anterior position on February 22. The patient was well. Abdominal palpation showed the fœtus to be lying transversely, head to the right, in the upper part of the abdomen, which appeared flattened above the umbilicus. The fœtal heart sounds were normal. The uterus could not be felt properly, although the abdominal wall was relaxed. No tenderness was present. The mother's condition was excellent. From February 22 to 25 the mother was well and walking about in the maternity ward. She mentioned that the baby was very active and kicking a lot in the upper part of the abdomen.

On February 25 the abdomen appeared flattened on top of its lower protuberance, and on palpation the uterus could not be felt and no uterine contractions could be induced by handling. The fœtus was easily palpable lying in its usual transverse manner, the head just above the right iliac fossa and the limbs palpable about the level of the umbilicus. The fœtal heart rate was 130 per minute. From the level of the umbilicus extending down into the pelvis was a firm cystic swelling dull on percussion. It occupied the centre of the lower part of the abdomen, with resonant areas in the iliac fossæ and flanks. The bladder was emptied by catheter and a pelvic examination made. The vulva and vagina were normal, the cervix was small and soft but closed. The uterus could not be felt. Occupying the pelvis and continuous with the abdominal tumour was a tense, cystic mass with the fœtus lying above all this. It could be moved so that the head approached the mid-line, but when it was let go it moved back to its position above the right iliac fossa.

The diagnosis was then considered to lie between the following conditions: (i) abdominal pregnancy, on account of the easily palpable fœtus, and of inability to palpate the uterus or uterine contractions; (ii) pregnancy complicated by a large ovarian cyst, which pressed on the lower part of the uterus and prevented entry of the fœtus into the pelvis. The second diagnosis was decided upon as being the more likely on account of the large cystic swelling in the pelvis and lower part of the abdomen and because of the rarity of the first condition. It was decided to attempt removal of the cyst, with Cæsarean section in the event of trouble.

The mother's pelvic measurements were large.

At 2 p.m. on February 28 under "open" ether anæsthesia the abdomen was opened by a mid-line incision from the pubis to the umbilicus. On opening the peritoneum a large reddish-blue cystic tumour was seen with pieces of material resembling coagulated pus stuck onto it. This tumour extended to the umbilicus and occupied the pelvis. Above it a loop of umbilical cord was seen. On holding up the abdominal wall, the child was seen lying transversely across the upper part of the abdomen, its head partly enveloped in a piece of ragged amnion, but the rest of its body and limbs mingling with the small intestine, transverse colon and omentum. The child was pulled out by the legs; it was cyanosed, but the cord pulsated strongly and in a few minutes it cried. The cord was then cut and the child was removed.

The abdomen was then examined. There was no free fluid at all, but about one ounce of *vernix caseosa* was found packed in the pouch of Douglas. Pieces of vernix were also scattered about the peritoneal cavity. The cystic tumour by this time had shrunk to about one-third of its original size and was seen to be the placenta contained within the left broad ligament.

The uterus was soft, congested and pushed to the right, and was the size of a three months' uterine pregnancy. The right Fallopian tube and ovary were normal. On the left side the tube was œdematous, enlarged and elongated to about eight inches. It ran along the top of the swelling in the broad ligament, but had a flattened centre third. The left ovary was slightly enlarged by a *corpus luteum* and lay in its normal position below the broad ligament.

The broad ligament was smooth. Its anterior peritoneal surface was normal. It bulged greatly and could be lifted out of the pelvis so that it had a broad, flat pedicle, in which could be seen and felt large arteries coming from the left side of the uterus. On its posterior surface midway between the tube above and the reflection of the peritoneum into the pouch of Douglas was a smooth round hole with the umbilical cord issuing from it. Around the point of exit of the cord from the broad ligament was a rolled-up cuff of amnion, the folds of which were stiff and hardened as if it had been rolled up for some considerable time. There were no adhesions of any kind in the pelvis or abdomen and no free fluid.

The left broad ligament, Fallopian tube and ovary were excised *en bloc* without hæmorrhage by clamping of the pedicle. The peritoneum of the lateral pelvic floor was sutured in a line, and the aodominal wall closed in the usual manner.

Convalescence of mother and child was uneventful. Lactation was satisfactory on the fourth day. The mother passed a decidual cast of the uterus on the eighth day after operation. She was out of bed and walking about on the fourteenth day.

The child was a female infant with no apparent abnormalities. She weighed four pounds two ounces at birth and was eighteen inches long. She cried well and seemed none the worse for her adventures. Expressed breast milk was given three-hourly at first. Later she was breast fed. Her birth weight had been regained by March 8, and she weighed seven pounds on April 8.

Examination of the Left Broad Ligament.

The anterior peritoneal surface was intact. On top of the broad ligament the œdematous uterine tube was eight inches long and patent throughout its whole length. When opened its floor seemed smooth and intact. Beneath it the broad ligament was occupied by a mass of spongy chorionic tissue and blood clot, with large vessels running into it from the direction of the uterine vessels on the aspect nearest the uterus. The ovary with its *corpus luteum* hung on the posterior surface of the broad ligament two inches below the tube, in what would have been its normal position if the ligament had not been stretched out. The peritoneum of the posterior surface of the broad ligament was intact except where it fused with the shrunken collar of amnion surrounding the smooth hole containing the cord. The cord was normal and sixteen inches in length (including the five inches left attached to the child on delivery).

The Mother's History.

In the days following the birth of the child a careful inquiry was made into the history of the mother, and the following information gathered. M.C.F. was born at Mackay on January 19, 1930. She had had the usual childish exanthemata. One and a half years previously she had tried to commit suicide with barbiturate tablets.

There was nothing of importance in the family history. Her menstrual periods began at the age of fourteen years, and were always regular, lasting seven days in every twenty-eight days. She had had no dysmenorrhœa. She began having intercourse with her future husband in March, 1946. Coitus occurred every second week until pregnancy was noticed. There was no history to suggest any venereal infection. The last menstrual period began on June 29 and finished on July 5, 1946. Morning sickness was first noticed late in July, 1946, and also swelling of the breasts. She was married in October, 1946. She had no recollection of the time when the first fœtal movements occurred. On October 7 she went to Innisfail Hospital to confirm the diagnosis of pregnancy. Examination *per vaginam* revealed the uterus to be the size of twelve weeks' pregnancy, and a small hard lump was felt on the right side of the uterus. The diagnosis of fibroid or bicornuate uterus was made. On October 12, after lifting a weight, the patient experienced severe pain in the left side of the lower part of the abdomen. On October 13 she was admitted to Innisfail Hospital because of the severe pain. There was no hæmorrhage *per vaginam*, no vomiting and no diarrhœa. Owing to retention of urine, her bladder had to be catheterized. The left side of the lower part of the abdomen was tender, and vaginal examination revealed the same findings as on October 7. In three days she was well and allowed to go home.

She attended the Innisfail antenatal clinic. On December 18 she had no complaints. The size of the fundus was that of a twenty-two weeks' pregnancy. The urine was clear. The blood pressure was 160 millimetres of mercury (systolic) and 80 millimetres (diastolic). On January 8, 1947, she was well. The fundus had enlarged to the size of a twenty-five weeks' pregnancy. The urine was clear. The systolic blood pressure was 120 millimetres of mercury and the diastolic pressure 80 millimetres. No abdominal palpation was carried out on these two occasions.

At the end of January, 1947, the patient travelled to Townsville by train. During the journey her abdomen became very painful, and the baby seemed to be moving about much higher up than before. She was then well until February 20, the date of admission to Townsville Hospital.

Comment.

Consideration of Mrs. M.C.F.'s complete story suggests the following sequence of events.

1. Pregnancy presumably occurred early in July, 1946, as symptoms of early pregnancy were present later in the same month. This gives the child an estimated age of between thirty-two and thirty-four weeks at delivery, corresponding to her size and weight.

2. Examination at Innisfail Hospital on October 7, 1946, disclosed a "uterus" the size of a twelve-weeks' pregnancy with a small hard lump on its right side. This lump must have been the uterus and the larger mass the ovum distending the middle of the left Fallopian tube, although rupture would be expected before that stage.

3. The attack of severe pain in the left lower quadrant of the abdomen would correspond to rupture of the ovum into the left broad ligament after exertion.

4. The occurrence of abdominal pain and freer fœtal movements on the train journey to Townsville suggest actual rupture of the sac or leakage of *liquor amnii*.

5. The sudden onset of abdominal pain on the night of February 19 and the morning of February 20 must have been due to rupture of the amniotic sac with irritation of the peritoneal cavity by *liquor amnii*. This would account for the events of the days from February 20 to 22. For eight days the fœtus was apparently free in the peritoneal cavity, because at operation the *liquor amnii* had gone and the remains of the amnion were tightly curled up round the base of the cord at its exit from the broad ligament. I cannot quote an authority on how long a fœtus could survive in the peritoneal cavity without its membranes and liquor surrounding it, but see no reason why it should not live provided its blood supply was intact and no undue pressure was exerted on it.

6. Examination of the left broad ligament, Fallopian tube and ovary showed that the ovum had been at some period within the broad ligament. It must have entered by rupture through the floor of the tube, though no lesion was found in the tube when it was opened. The posterior layer of the left broad ligament then bulged out upwards and backwards without stripping peritoneum from the floor of the pelvis, as this peritoneum was in its normal position at operation.

7. The complete absence of adhesions is against any variety of abdominal implantation of the ovum other than an intraligamentary one.

8. This pregnancy can then be regarded as a secondary abdominal (intraligamentary) pregnancy with secondary rupture into the peritoneal cavity. There is no obvious reason why this condition should have developed, as the mother's history contains nothing to suggest previous pelvic trouble.

Survival of the fœtus in such a pregnancy must be rare, and in this case, in which the correct diagnosis was missed, the child was very fortunate indeed.

Acknowledgement.

I wish to thank Dr. N. Aroney for his notes on the attendances of Mrs. M.C.F. at Innisfail Hospital.

Bibliography.

T. W. Eden and C. Lockyer: "Gynæcology for Students and Practitioners", Fourth Edition, page 216.

Reviews.

BOVINE TUBERCULOSIS.

In the monograph "Bovine Tuberculosis", with the subtitle "Including a Contrast with Human Tuberculosis", John Francis, of the Biological Laboratories of Imperial Chemical Industries, Limited, has compiled a searching and concise review of all major problems inherent in the subject.[1] There is naturally much discussion of the relative extent of tuberculosis among cattle in different countries, and the measures which have been adopted for the detection and eradication of the disease.

The morbid anatomy of tuberculosis in bovines is well presented, in a series of fourteen protocols, pertaining to which are thirty-one excellent photographic illustrations; this section has the further merit that it is based on the author's own careful observation.

In comparing and contrasting tuberculous processes as they occur in cattle and in the human subject, the author gives full consideration to current views on the pathogenesis of the human disease as propounded by Pagel, Terplan and Rich. It has been customary for many years to consider tuberculosis in man as divided broadly into "childhood" or primary infection, and "adult" or reinfection types. It is now more than doubtful whether this distinction can be sustained, but if it is allowed to stand for the present, it may be said that in the human subject, many primary infections of the lung heal completely, especially those acquired in childhood. Francis shows that the disease is different in bovines, in which animals there is little or no evidence that there is any difference in susceptibility at various ages. Further, the complete healing of lung lesions is much less common in cattle than in man, and bronchogenic spread usually occurs; the process may be very chronic, but it follows that nearly all tuberculin reactors in a herd of cattle are infective for others. The behaviour of the disease in cattle, therefore, would appear to parallel closely that prone to be followed by "late primary infection" or primary infection in the adult human subject. The author also makes the point that chronic bronchogenic tuberculosis in man usually follows reinfection and does not involve the regional lymph nodes, but in the cow, whether this condition is associated with reinoculation or simple primary infection, large caseous lesions are usually found in the related lymph nodes.

In the study of bovine tuberculosis veterinary science is in the position of advantage that there is no obstacle to the application of direct experiment, and by such method the interesting fact has been established that cattle are much more susceptible to aerogenous than to alimentary infection.

[1] "Bovine Tuberculosis, including a Contrast with Human Tuberculosis", by John Francis, B.Sc., M.R.C.V.S.; 1947. New York, Toronto and London: Staples Press, Limited. 8½" × 5½", pp. 220, with many illustrations, some of them coloured. Price: 25s.

According to the author of this monograph 80% to 90% of all tuberculous cattle acquire infection via the air passages.

It is disappointing to learn that in a field in which large-scale controlled experiment should not be handicapped by the difficulties which attend it in the study of human tuberculosis, the value of vaccination as a means of reducing the incidence of tuberculosis has not been convincingly demonstrated. A striking immunity can be induced in cattle by vaccination with *Bacille Calmette Guérin* ("B.C.G.") or the vole bacillus vaccine, but such immunity wanes after a few months, and apparently cannot be sustained by revaccination. Controlled trials of the prophylactic value of "B.C.G." vaccination carried out under semi-natural conditions have given disappointing results.

The book contains a wealth of information, on the whole well arranged, in the comparatively small space of 187 pages. There is more than one suggestion of hurried proof-reading, and at the beginning of the last paragraph on page 130 is to be found what is either a serious omission or an attempt at the impossible feat of constructing a sentence without a verb.

Commendable features are the abstracts appended to the several sections of the book, and the concluding epitome of the whole of the subject matter traversed. Last, but by no means least, is the exhaustive bibliography. Four hundred references attest the industry and tenacity of purpose of the author, and in themselves render the monograph worthy of acquisition by students of veterinary science and public health officers.

MENTAL HEALTH.

DR. J. H. EWEN, lecturer in psychological medicine, Westminster Hospital, has written "Mental Health", described as a practical guide to the disorders of the mind.[1] Dr. C. Friedman assists with a chapter on special treatments.

In this small book the author sets out to cover in outline the subject matter usually found in a much larger textbook of psychiatry. The first sixty pages of the book comprise short sections of psychopathology, ætiology, symptoms and technique of examination. Following this the psychotic and emotional disorders are dealt with on much the same plan as in standard British textbooks. The final section of the book, dealing with legal aspects of mental disorder and of certain administrative responsibilities in mental hospital practice, will be found useful mainly by physicians undertaking mental hospital practice in Great Britain.

The value of this book does not rest upon any new or unusual material, but rather on the fact that it is easy to read and systematically set out. Although the matter is of necessity condensed to a mere outline, it possesses the clarity and continuity of a much larger work.

The section devoted to the neuroses will not be as helpful an introduction to the student as the preceding section on the psychoses, as it is almost impossible to cover in part of a small volume the whole range of emotional disorder, its basic mechanisms, clinical manifestations and management.

The authors deserve special commendation for the way in which various standard complex psychological theories are summarized with a minimum of jargon, so that they can be understood readily by the average medical student, and this book can be recommended to students approaching for the first time the study of mental disorder.

MAN AND DISEASE.

IN "Medicine", Volume I, by A. E. Clark-Kennedy, physician to the London Hospital and Dean of the Medical School, a refreshing and urgently needed approach to the study of medicine is presented.[2]

It is not a book in which the clinical features of disease are described, but rather one that correlates the many branches along which medical science has progressed. The extreme complexity of the human body and mind governed by hereditary and environmental factors is presented so clearly through successive chapters that medical student and graduate alike could not help but be stimulated by this book. At the same time every student of medicine will gain from it a greater understanding of the patient and his disease.

The volume is divided into six chapters, each one complete in itself. The first deals with the integration of the pre-clinical subjects, namely, energy and matter, life, organic evolution, heredity, development, constitution, consciousness and mind. The next two give a full and interesting account of symptoms and signs interpreted in terms of alteration of structure or disturbance of function of the body or mind. The last three chapters require closer concentration and adopt a philosophical approach in describing the hereditary and environmental risks to which man is exposed, the reaction of body and mind to different forms of noxious internal and external stimuli, and lastly the nature of disease in so far as it affects structure and function.

On the nature of disease one is presented with a most logical discussion on the seven pathological processes of the body, namely, congenital defects, mechanical injury, nutritional deficiency, chemical poisoning, acute and chronic infection, newgrowths, and degenerative changes of advancing age. The effect of physico-chemical changes in bringing about these pathological processes is described and the theme develops naturally into the study of the course of changes in structure and function of the human body. The volume closes with a philosophical discussion of the real nature of disease, and here the author has integrated superbly, organic, functional and psychoneurotic disorders.

The continuity of the subject matter is so simply phrased that the reader is not aware of its underlying significance until the summaries at the end of each chapter are read.

No one without a vast knowledge of the field of human experience could integrate so well the medical sciences in such a small space, and those who read the volume will return to it many times to gain further knowledge and mental stimulation.

We look forward to the second volume on diagnosis, prevention and treatment.

MODERN DEVELOPMENT OF CHEMOTHERAPY.

"MODERN DEVELOPMENT OF CHEMOTHERAPY", by E. Havinga, H. W. Julius, H. Veldstra and K. C. Winkler, is one of a series of monographs on the progress of research in Holland during the recent war.[1] In a foreword the editors of the series, Dr. R. Houwink and Dr. J. A. Ketelaar, state that the publication of these monographs was planned in the first years of the war as a token of the undaunted spirit of the Netherlands. The purpose was to show the world that scientists in the Netherlands had remained active during the five years of German occupation. As it was the editors aim to present these monographs as early as possible after the war. The majority of them were prepared under war conditions without "exhaustive reference to the Anglo-Saxon literature" which was not available in territory under German occupation. The editors state that during the war research was continued extensively in all directions in spite of the ever-growing burden of oppression and starvation. One cannot but admire the spirit of the scientists who continued to work in the face of such difficulties; at the same time it is obvious that they were severely handicapped by their isolation. It is essential for any research worker to have access to world literature, otherwise he is liable to be working in a blind alley, cut off from the highroads of human progress. The loss, of course, was not only on one side; if Holland was cut off from work being done elsewhere, British and American workers were equally deprived of the help that Dutch scientists might have given them. Two main lines of research are described in this book; the first is the mechanism of the action of sulphonamides and of para-aminobenzoic acid; the second is represented by a series of chemical investigations, including the synthesis and activity of sulphanilamide derivatives and of related compounds. There is no mention of sulphadiazine or of other modern sulphonamides. As the writers themselves observe, "some of the older series of experiments recorded here now seem to be overtaken; either by the researches of workers abroad or by our own work". This book contains 175 pages measuring 5½ by 8 inches. In a foreword, the editors express their greatest admiration for the publishing house of Elsevier, which took very serious risk in preparing this series in wartime, when all activity on behalf of such international purposes was strictly forbidden.

[1] "Mental Health: A Practical Guide to Disorders of the Mind", by John H. Ewen, F.R.C.P.E., D.P.M., with a chapter on "Special Treatments and their Practical Technique", by C. Friedman, M.D. (Vienna), L.R.C.P. and S. (Edinburgh); 1947. London: Edward Arnold and Company. 8½" × 5½", pp. 270. Price: 12s. 6d.

[2] "Medicine", by A. E. Clark-Kennedy, M.D., F.R.C.P.; Volume I: The Patient and his Disease; 1947. Edinburgh: E. and S. Livingstone, Limited. 9½" × 6", pp. 396. Price: 20s.

[1] "Modern Development of Chemotherapy (Monographs on the Progress of Research in Holland during the War)", by E. Havinga, H. W. Julius, H. Veldstra and K. C. Winkler; 1946. New York and Amsterdam: Elsevier Publishing Company, Incorporated. London: Cleaver-Hume Press, Limited. 8" × 5½", pp. 190. Price: 15s.

The Medical Journal of Australia

SATURDAY, MARCH 27, 1948.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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GROUP PRACTICE.

DURING the last few years group practice has been mentioned in medico-political discussions from time to time as one of the means by which the present method of medical practice may be made suitable for the changing needs of the present time. It is true that no reference has been made to it lately, but it is still in the list of additions and changes mentioned in the Federal Council's statement of policy. The subject was discussed at some length in these columns in May, 1943. On that occasion reference was made to the interim report of the Medical Planning Commission issued in Great Britain. (This report, it may be remembered, was printed in the *British Medical Journal*, but all copies of the issue containing it were lost on the way to Australia as a result of enemy action. The report was therefore by special permission published in this journal.) In addition, the main features of a brochure on group practice prepared by the Bureau of Economics of the American Medical Association were set out in some detail. The information in the brochure was based on replies to a *questionnaire* received from 266 secretaries of county medical societies dealing with more than 500 groups. It will be remembered that the Medical Planning Commission mentioned three types of group. The first was that of a health centre scheme such as would exist in a well-planned whole-time salaried service. The second type concerned a group of general practitioners working at a centre rented or owned by them. The third was the continuation of the partnership system in a health centre plan, the centre being regarded simply as a communal partnership surgery. It was pointed out that the first type of group practice was out of court because of the fact that a salaried service was unacceptable to the Federal Council, and readers were referred to the Federal Council's report on group practice published in this journal in the issue of April 24, 1943. In our discussion on May 15, 1943, it was pointed out that the observations recorded in the American brochure would not necessarily apply to Australian conditions and Australian practitioners. The general impression conveyed by the American observations was not particularly encouraging. For example, in only a minority of instances had it been possible to assemble a body of specialists and adequate scientific equipment such as would constitute a fairly comprehensive medical unit, capable of offering a complete medical service. This subject, which in ordinary circumstances would not be brought forward at present for discussion, is mentioned so that medical practitioners may be informed of the latest news about medical group practice in the United States.

G. Halsey Hunt, Senior Surgeon in the United States Public Health Service, and Marcus S. Goldstein have reported on a *questionnaire* survey made in 1946.[1] Questions were sent to groups whose names appeared in the lists of various bodies; the total number was 981. The *questionnaire* was issued in the summer of 1946 and by the end of the year replies had been received from 774 (78·9%). It was found that only 368 groups were suitable for study. Among the groups excluded from the study were those with fewer than three full-time practitioners, those in which a single specialty was practised, those consisting of organized hospital staffs where the practitioners participated on a part-time voluntary basis, and those in which members shared overhead expenses but carried on medical practice as individuals. Among the groups included were those comprising practitioners employed by industrial companies, hospitals, consumer cooperatives or individual owners, provided the practitioners carried on their principal professional activity by working together as a group. Of the 368 groups forming the basis of the report six were industrial and two were consumer cooperative groups providing medical care only for the employees of single companies. Thirty-six single specialty groups were not included because they represented a "quantitative rather than a qualitative increase over the work of an individual practitioner". It is interesting to note that during the last few years some groups have been disbanded (the disbanding is described as the mortality of medical groups). The disbanding of groups was discovered by the American Medical Association. The list of groups used by the Association in 1940 was used again in the present survey. Of 334 *questionnaires* sent out 282 were returned; 19 groups had been disbanded and 36 others had apparently become informal associations. This means that at least 19·5% of the groups which were active in 1940 (some of the 52 which did not return the present *questionnaire* may have been disbanded) had disbanded or had ceased to operate as groups during the period 1940 to 1946. Unfortunately there is no information about the reasons for disbanding, information which might throw some light on the whole subject.

When the available information was dissected it was found that nearly half of the groups were located in the North Central States and that more than half (58%) were in cities with a population of less than 25,000; no less than 82% were in communities of less than 100,000 persons. These facts are in essential agreement with the findings of a survey made by the American Medical Association in 1940. The size of the groups varied a good deal. Groups of three to five practitioners made up 53% of the total number and 80% had ten or less than ten practitioners. There were more than 15 groups which

[1] *The Journal of the American Medical Association*, December 6, 1947.

had as members more than twenty practitioners. The median size of all the groups was 4·7 full-time practitioners. Of the total of 3084 full-time practitioners in the 368 groups 48·5% were in groups of ten or less; 51·5% were in groups containing 11 or more full-time practitioners. Of the 368 groups, 93 (25%) had part-time practitioners on their staffs. The average number of part-time practitioners in these 93 groups was 1·7. Only three groups had more than 12 part-time members. As might be expected, there was a tendency for smaller groups to be found in the smaller cities and larger groups in the larger cities. At the same time some 33% of the large groups (those with 11 or more full-time members) were found in communities of less than 25,000 persons. The primary activity of 349 groups was stated to be the provision of general medical care; 19 groups gave service by consultation or treated patients referred to them by other practitioners. Over three-quarters of the groups were partnerships and most of these also employed additional practitioners. Almost 10% of the groups were owned by one member who employed the others in the group. One point that should be noted is that in 8·4% of the groups all the practitioners were employed by a sponsoring organization. Full information about these groups would be welcome. We read that they are found more frequently in cities with the larger populations. These groups also tend to be large and have more part-time men on their staffs than groups organized in any other way. Of the 368 groups 361 noted their range of practice. Almost all covered the fields of medicine and surgery; a decreasing number dealt with obstetrics, radiological examinations, gynæcology, pædiatrics, oto-laryngology, ophthalmology and dentistry. Of the special fields named the average number furnished by all 361 groups was 6·8.

Questions of hospital relations, administration and so on remain to be mentioned. The question was asked whether the group had its "own hospital". Of the 368 groups, 117, or 31·8%, replied in the affirmative. In 1940 the number of groups in the American Medical Association survey with hospital ownership was 25%. The percentage of 31·8 is high, but it should be noted that the question was so framed that it could not be certain that an affirmative reply meant actual ownership or integral affiliation without ownership. Hunt and Goldstein, however, have a strong impression, gained from sources outside the *questionnaire*, that actual ownership is meant. It is also perhaps important that the groups owning hospitals comprised a higher percentage of those with a sponsoring organization than of those organized in any other way. The size of the hospitals, according to 114 groups replying to the question, ranged between six and 350 beds with an average of 72. One-fourth of the groups had hospitals of 100 beds or over. In regard to administration of the groups, 218 of the 368 had a medical director; 123 had no medical director and from 27 groups no reply to the relevant question was obtained. Of the total number of groups 292 had a "business manager"; 69 groups were stated to have no business manager, and no information on the point was given by seven groups. Much of the importance of these replies is lost because there is no universally accepted definition of the term business manager; sometimes the term is applied to secretaries or bookkeepers who have no real managerial functions. Of the 368 groups, 344 replied to a question about the employment of graduate nurses in the clinic; 301 or 88% employed graduate nurses. The total number of clinic nurses was 1469, or approximately one graduate nurse to every two full-time medical practitioners. A medical prepayment plan was operated by about 15% of the groups, and, as might be expected, was found to be relatively more frequent among the large than among the small groups.

From the account that has been given of Hunt and Goldstein's survey it will be seen that the group practice in the United States is probably more extensive than it was in 1943; the picture is more "variegated" than it was five years ago. Clearly, as has been remarked on previous occasions, group practice is an expensive business. We do not propose to comment further on the results of the survey; the facts can tell their own story. It is a subject which should interest many Australian practitioners; it is to be hoped that those with access to the report will study the complete document.

Current Comment.

PROGNOSIS IN BREAST CANCER.

CANCER of the breast is still the commonest form of cancer in the human subject and therefore a target of great importance in the campaign against malignant disease. There are, however, grounds for encouragement in this particular field in regard to the results of treatment. A particularly complete follow-up study has been made by Arthur B. McGraw[1] of patients who have undergone radical mastectomy of the Halsted type at the Henry Ford Hospital during the thirty years prior to 1941 and who have survived operation for at least five years. During the period of thirty years the operation was performed 412 times with a mortality rate of 2·2%. All but six patients were followed up and it was found that 177 (43%) survived the operation five years or more. Of this group of 177 patients, 48 either died of metastases or were living with recurrence; a quarter of these lived for over ten years; in more than one-half there were no signs of recurrence for over five years, and in about one-sixth for over ten years. Careful scrutiny did not provide any significant information relating to the instances of delayed recurrence, but it is notable that nearly two-thirds of the subgroup were found to have the axillary lymph glands involved at the time of operation. The next subgroup investigated of the 177 five-year survivors were those who died from intercurrent infection. There were 29 in this group, of whom over half lived for more than ten years; there were many older women and McGraw points out that, if they had not had the advantage of radical surgery, they would have died much sooner than they did and of their cancer. In the third subgroup, those who were living and well five years or more after operation, there were 100 patients; more than one-half had survived for over ten years and 12 for over twenty years. The most significant factor amongst this last subgroup was that only 30 of the 100 patients were observed at operation to have metastases in the axillary lymph glands. Of those in the subgroup who had survived for over twenty years, one-half had been operated on less than one month after noticing their tumours and two-thirds were free of axillary involvement; it is worth noting, however, that of these 12 twenty-year survivors six had scirrhous carcinoma, four adenocarcinoma, and one medullary carcinoma, and eight had some combination

[1] *Archives of Surgery*, September, 1947.

of retraction of the skin, retraction of the nipple or fixation of the growth to the thoracic wall. A further point brought out by this study was that the practice of removing tumours of clinically uncertain diagnosis for immediate examination before a decision was made on radical mastectomy had not apparently had any adverse effect.

In the discussion that followed McGraw's paper, W. D. Gatch drew attention to the importance in prognosis of the involvement or otherwise of the axillary glands at operation and also to the impossibility of ever being sure that recurrence would not take place; he had seen recurrence seventeen years after operation and abdominal metastases had been found *post mortem* forty-three years after radical mastectomy. S. W. Harrington, of the Mayo Clinic, also stressed the significance in prognosis of axillary lymph gland involvement and quoted a study of over six thousand patients to show that for the increasing survival periods of patients without metastasis to the axillary glands, the rates showed progressive improvement as compared with the rates for those with axillary metastasis.

The importance of axillary lymph glandular involvement has been emphasized also by Herbert C. Chase[1] in a recent general discussion of breast cancer. Chase strongly advocates the most radical type of operation and does not consider adequate the Halsted operation employed throughout McGraw's series. He is particularly concerned that all groups of lymph glands should be removed and that the implantation of cancer cells in the operation field should be avoided; to ensure the destruction of implants he advocates X-ray therapy, but makes it clear that X-ray therapy will not compensate for the incomplete removal of axillary lymph glands. Some further points brought out by Chase and bearing on prognosis are that, although the incidence of carcinoma of the breast is increasing in the United States (nearly 1% *per annum*), the cure rate is increasing more rapidly than the incidence. Of the 15,000 women who die of cancer of the breast each year in the United States, it is considered that 10,000 could be saved by surgery alone if the lesion was discovered while confined to the breast; after the disease has spread to the axilla only 6000 can be saved by surgery, X-rays and radium combined. Chase discusses at some length various factors which may be concerned in the ætiology of breast cancer, but there is too little known in this regard for the disease to be in any way preventible. The hope of further progress in controlling breast cancer lies in continued education of the public and of medical practitioners in the early certain diagnosis of breast abnormalities and in "the education of surgeons towards a more thorough, extensive, and meticulous radical operation".

PERSONALITY CHANGE AFTER PREFRONTAL LEUCOTOMY.

THE operation of prefrontal leucotomy has been discussed on a number of occasions in these columns and was the subject of a series of articles published in the issue of October 25, 1947. There had not, however, been sufficient time to allow of any comment based on observations over more than a short period after operation. Reference was made on January 26, 1946, to work being carried out at Dumfries, and it is now of interest to read a report[2] by L. Frankl and W. Mayer-Gross of the further progress of some of the earlier Dumfries patients. Of 170 patients operated on between February, 1943, and June, 1946, 77 were discharged from hospital; nine were readmitted to hospital and this report is concerned with the other 68. Of the 68, 14 had lived at home for more than two years, 28 for between one and two years, and 26 for less than one year; none had been at home for less than six months. More than half of the 68 (who without the operation would have been chronic invalids) now earn their living and more than three-quarters are usefully employed. By far the best results have been among professional people and housewives. It is appreciated that these are much smaller proportions of the 170 patients operated on, but the purpose of this report is not so much to discuss the relative incidence of success and failure as to assess the effect produced in successful and relatively successful cases and to apply these findings in the selection of patients for operation. Reports on individual patients are given and are of much interest, but it is sufficient here to quote the summing up by Frankl and Mayer-Gross of the character of a patient after successful leucotomy.

The patient is physically healthy, a good eater and sleeper, with considerable euphoria. His prevailing mood is cheerfulness; he does not worry, is happy and contented, and shows it. He has a high opinion of himself and his abilities. He may take his responsibilities too easily or may shirk them altogether. He leads an active life, is restless, and likes movement and change. His interests and hobbies are varied and variable, with a preference for light entertainment and superficial pastimes. He is easy-going, a good mixer, and fond of social life and of being on good terms with everybody.

On the other hand, his relations with the people round him are often without depth of feeling. He shows little sympathy or consideration for his next of kin or anybody else. He neither regrets nor repents for whatever he may have done or said. He may be self-willed, headstrong and unable to see the other person's point of view. He tends to quickly passing outbursts of temper. He may attend to his work as before or may do less well, but he invariably thinks he does well; he is not a perfectionist.

The individual picture is modified by personality traits present before operation, but in most cases features such as obsessions, rituals, guilt feelings and anxiety, even if persistent, have lost their predominant influence. It is interesting to note that, though the picture presented above has many of the features of the schizophrenic personality, these "schizophrenic" traits occurred just as frequently amongst those who had not previously had a schizophrenic disorder as amongst those who had. Frankl and Mayer-Gross point out the close resemblance between this personality picture and that resulting from frontal lobe damage after head injury or lobectomy. They suggest that "the operation, if successful, provides the patient with a new framework of personality caused by the isolation of his pre-frontal areas". The reconstruction of the patient's mental life on this new basis will naturally be slow, and some of the patients were still improving two years after operation.

The conclusions reached bear first on the selection of patients; the operation has been worth while for those with severe obsessional states, with chronic affective psychoses and with schizophrenia, apart from the hebephrenics, whose condition is too akin to the frontal lobe syndrome to permit improvement. In selecting the time of operation, it is considered that rebuilding of the personality on the new basis is always possible so long as one can be certain that "emotional and intellectual life are preserved behind the screen of psychotic behaviour"; but leucotomy is not in most cases necessary in the early stages of illness and the usual less drastic therapies should be tried first and spontaneous remission waited for, if it is at all likely to occur. As to operative procedure, the theory of these investigators implies that the basis of operation is the production of enough frontal lobe symptoms to combat the symptoms of the psychosis; this aspect, however, requires much more clarification. Finally, it is pointed out that the life and personality of the patient after operation are pliable and ready for systematic reconstruction; he should not be discharged from hospital too early, rehabilitation facilities must be adequate, and both relatives and family doctor must be fully aware of what is required of them.

This report makes no sweeping claims. The authors put forward a theory which is treated as tentative only, but nevertheless provides a useful working basis. The significant part of the paper is the picture presented of the personality seen after leucotomy, based on a reasonable period of observation. Its general trend favours the conservative attitude towards the operation and suggests that further observations are likely to confirm this attitude.

[1] *Surgery, Gynecology and Obstetrics*, December, 1947.
[2] *The Lancet*, December 6, 1947.

Abstracts from Medical Literature.

GYNÆCOLOGY.

The Rh Factor in Abortion.

ARTHUR B. HUNT (*American Journal of Obstetrics and Gynecology*, March, 1947) has made a study of 93 patients with recurrent abortion with a view to assessing the importance of the Rh factor in the ætiology of this condition. He states that if absence of the Rh factor plays a part in the causation of abortion it is overshadowed by other causes and is not apparent statistically. In the present state of our knowledge the opinion that the prognosis for pregnancy is poor in an Rh-negative woman married to an Rh-positive husband is not warranted. She should receive the same investigation and treatment as is given to any group of relatively infertile patients and her chances for successful termination of pregnancy are worth an effort on the part of the obstetrician. The Rh factor possibly has been over-emphasized as a cause of abortion and miscarriage at the expense of more common causes, such as dysfunction of the ovaries, pituitary body, thyreoid glands, and possibly the testes of the husband.

The Fibrous Nature of the Human Cervix and its Relation to the Isthmic Segment in Gravid and Non-Gravid Uteri.

D. N. DANFORTH (*American Journal of Obstetrics and Gynecology*, April, 1947) reports a study of the cervix and isthmic segment of the uterus from 12 pregnant uteri and 46 non-pregnant uteri removed surgically, and considers that certain fallacies exist in our present understanding of the anatomical and physiological significance of the isthmus. An explanation on anatomical grounds is offered for certain cervical and isthmic phenomena of pregnancy which have not been fully understood. In the opinion of the author, from functional and anatomical standpoints the uterus should be considered as being composed of two major parts—the cervix and the corpus—according to whether the essential tissue is fibrous or muscular. The concept of the *isthmus uteri* as a separate entity should be eliminated.

The Technique of Total Hysterectomy.

GEORGE E. JUDD (*The Western Journal of Surgery, Obstetrics and Gynecology*, April, 1947) reviews the development of operative technique in total hysterectomy over the past seventy years and stresses the importance of reconstruction of the essential supports of the pelvic floor after removal of the cervix. The combination of total abdominal hysterectomy and anterior colporrhaphy has always offered technical difficulties, because so many operations for cystocele are based on the advancement of the bladder upon the uterus. Vaginal hysterectomy has produced excellent results where there has been a pathological condition of the uterus or adnexa combined with prolapse, but this operation is not always possible on account of the size of the uterus, the presence of adhesions or associated involvement of the adnexa. The gynæcologist should deal with all pathological conditions of the pelvic structures in a single operative session when possible. The author describes in detail his technique of total abdominal hysterectomy with combined reconstruction of the continuity of the upper pelvic floor.

Primary Ovarian Carcinoma.

JAMES V. CAMPBELL AND DAVID SINGMAN (*The Western Journal of Surgery, Obstetrics and Gynecology*, May, 1947) report a study of 69 patients with primary carcinoma of the ovary, which has been classified as cystic, solid or functional. Cystic tumours predominated, by far the greatest number being papillary serous cystadenocarcinoma; an 8% five-year survival rate occurred among 51 patients with cystic malignant ovarian disease. Solid carcinoma was present in 11 of the 69 patients; the five-year survival figure for these was 30%. Malignant functional tumours studied included two cases of granulosa-cell carcinoma and one of theca-cell carcinoma; the three patients have lived beyond five years, but one patient with granulosa-cell carcinoma died at the sixth post-operative year. Two-thirds of all patients with primary ovarian cancer are in the forty to sixty years age group, one-quarter are over sixty years of age, and one-tenth are under forty years of age. The presence of metastases and/or ascites is important in prognosis and is associated with a greatly reduced survival time. No conclusions could be drawn as to the effects of X-ray therapy. The higher the grade of malignancy by histological classification, the less chance has the patient for survival. The onset of ovarian malignancy is insidious and the first signs or symptoms recognized by the patient frequently occur when the condition is already inoperable. In the series of patients studied there was an over-all five-year survival rate of only 14%.

Total Hysterectomy.

EQUINN WILLIAM MUNNELL (*American Journal of Obstetrics and Gynecology*, July, 1947) discusses subtotal and total hysterectomy on an analysis of 1798 patients of whom 1583 underwent the subtotal operation and 215 total hysterectomy. The operative mortality rate in the group of 215 total hysterectomies performed for malignant and benign conditions was 2·32% as compared with a mortality rate of 1·76% for the 1583 subtotal hysterectomies. The outstanding causes of death after total hysterectomy were pulmonary emboli and cardio-vascular accidents. Among post-operative complications urinary tract infection was about the same in both groups, parametritis and cellulitis were less frequent after total hysterectomy, and pulmonary embolus was more frequent after total hysterectomy. Post-operative catheterization was necessary more frequently after subtotal operation. There was no significant difference in post-operative distension for the two operations. There is general agreement that total hysterectomy is to be preferred as a routine operation by the skilled operator. The proper selection of cases involves the ease with which the operation may be performed, the skill of the operator, the patient's general condition, the degree of fixation of the uterus, the presence of adhesions and the obesity of the patient. Prevention of cancer of the cervical stump is an adequate reason for performing total hysterectomy in the presence of benign lesions so long as the operation does not introduce an element of extra danger to the patient. Following a study of 64 patients who had either total hysterectomy or subtotal hysterectomy during an active sexual life, the author is of the opinion that the presence of the cervix has nothing to do with the production of orgasm in the female; the same can be said about the ovary and the body of the uterus. Cancer of the cervix can develop in a nulliparous cervix which at one time was healthy. Conversely, most cervices with cervicitis, lacerations and erosions never develop cancer. It is important that the decision for or against total hysterectomy with a proved benign condition of the cervix should be made more on the factor of ease of operation than on whether or not the cervix is clean.

Vaginal Hysterectomy.

HOWARD C. STEARNS (*The Western Journal of Surgery, Obstetrics and Gynecology*, April, 1947) stresses the importance of the cardinal ligaments, the utero-sacral ligaments and the vesico-vaginal fascia in vaginal hysterectomy and describes his technique which utilizes these supports. The mortality rate among 292 patients submitted to operation was 1·02% and all deaths were due to post-operative bronchopneumonia. In the author's opinion vaginal hysterectomy is a dependable and valuable procedure, and in many conditions of prolapse no substitute operation can take its place.

The Prevention of Gynæcological Cancer.

ROBERT J. CROSSEN (*American Journal of Obstetrics and Gynecology*, August, 1947) states that the prevention of carcinoma in the ovaries, uterus and external genitals is made possible by the recognition of two common conditions as predisposing factors, namely, chronic irritation and involution changes. Reduction in the incidence and mortality of cancer of the cervix has followed educational publicity concerning early signs and symptoms, the performance of routine gynæcological examinations, and increasing recognition of the importance of surgical treatment for chronic cervicitis. In the treatment of chronic cervicitis the affected tissue must be removed in a way which permits thorough microscopic check. Destruction of tissue by cauterization or coagulation precludes this and invites the risk of overlooking early cancer. The author states that "conization" is the method of choice and, if facilities are not available for this procedure, conical excision with a knife is recommended. In the performance of hysterectomies the cervix should be removed, but if there is some contraindication to total hysterectomy the cervix should be "coned". *Leucoplakia vulvæ* affords an example of chronic irritation which is closely related to subsequent cancer. The author agrees that the incidence of vulval cancer might possibly be halved if complete vulvectomy was adopted for the treatment of well-developed *leucoplakia vulvæ*. The ovaries and the uterus are temporary organs; after the menopause their function has ceased and they are involuting organs on their

way out of the active economy. Then they not only are functionless, but carry the special menace of involuting structures which is a predisposition to cancer development. Primary ovarian carcinoma is the most insidious of malignant diseases of the pelvic organs and the one most frequently overlooked until it has reached a hopeless stage. Three principles of treatment are necessary in reducing deaths from this disease: removal of the involuting ovaries whenever the abdomen is opened under circumstances which permit such removal; the insistence on regular pelvic examinations at six-monthly intervals; the utilization of any opportunity for thorough vaginal examination under anæsthesia. In the treatment of the involuting uterus for benign pathological conditions a careful assessment in the individual of three important factors is necessary: firstly, the chance of satisfactory relief by radiation; secondly, the risk of the later development of cancer; thirdly, the risk of a major operation. With an experienced surgeon the average mortality risk from removal of the uterus and ovaries is 1% to 2%. Without radium treatment the malignancy risk for uterus and ovaries is 2·38%, and with radium treatment the risk is 0·89%. The author considers that total hysterectomy with bilateral oophorectomy is the treatment of choice for climacteric patients with troublesome uterine myomata provided that the operative risk is good. In patients handicapped by a high operative risk, radiation preceded by diagnostic curettage and conization of the cervix (when necessary) is a life-saving measure and strongly indicated. Prolongation of the menopause does not imply "renewal of youth" and the prolonged action of endogenous œstrogen is apparently an important factor in the ætiology of *carcinoma corporis uteri*. Bleeding beyond the usual age does not call for œstrogen medication, but for investigation to determine whether endometrial carcinoma has already commenced.

OBSTETRICS.

Studies in Prematurity, Stillbirth and Neonatal Death.

C. M. DRILLIEN (*The Journal of Obstetrics and Gynæcology of the British Empire*, August, 1947) continues her statistical analysis of 7599 births occurring in the Simpson Memorial Pavilion, Edinburgh, from 1942 to 1945 inclusive. The delivery of *primiparæ* was by means of forceps three times as often as that of *multiparæ*. For mature babies instrumental delivery was more common than for premature, but the Cæsarean section rate among the latter was much higher—8·7% as against 3% for mature babies. A consideration of the outcome according to the mode of delivery suggested the following conclusions: (i) Instrumental delivery appears to be as safe as spontaneous vertex delivery for premature infants, though it carries an increased risk (2·1%) in the mature. (ii) Breech delivery has a considerably greater wastage rate than any other method in both premature (52·8%) and mature (15·1%) births. (iii) There is an increased risk in Cæsarean section for both premature (2·9%) and mature (4·4%) infants. When cases of hydramnios, *placenta prævia* and prolapsed cord were excluded, there was still a definite risk to the fœtus in cases of breech delivery, greater for mature than premature infants; the survival rate was 17·6% less than in cases of spontaneous vertex delivery. In the analysis of Cæsarean sections the only significant statistical finding was that emergency Cæsarean sections for prolonged labour with contracted pelvis, uterine inertia, "failed forceps", severe toxæmia, *placenta prævia*, ruptured uterus and other causes carried a definite risk (8·3%) compared with spontaneous vertex delivery. The incidence of spontaneous vertex delivery fell greatly with increasing maternal age, whilst that of instrumental delivery and Cæsarean section rose. There were 204 neonatal deaths—142 in premature and 62 in mature infants, and 373 stillbirths—177 in premature and 196 in mature or post-mature infants. Nearly one-quarter of the "premature" deaths took place in the first six hours and one-half on the first day. Of the "mature" deaths 25% occurred during the first day. Prematurity alone, asphyxia and pneumonia were the commonest causes of death in premature infants; congenital defects accounted for the majority of deaths in mature infants. Death rates are higher for males than for females in all but the congenital defect group. There was a rise in the death and stillbirth rate from congenital defects with increasing maternal age. Intracranial hæmorrhage was found in 65 out of the 204 infants who died.

Veratrum Viride.

FREDERICK C. IRVING (*American Journal of Obstetrics and Gynecology*, November, 1947) reviews the pharmacology of veratrum alkaloids of which *Veratrum viride* is the most outstanding because from its rhizomes is obtained "Veratrone". The administration of the drug produces a slowing of the pulse and a fall in blood pressure from a reflex stimulation of the vagus arc. It also has an anticonvulsant action probably secondary to the vasodilatation which occurs. Toxic symptoms are vomiting, burning sensations in the mouth, throat and stomach, profuse sweating, giddiness, headache, and in some cases relaxation of the vesical and anal sphincters. From 1940 to 1946, 32 eclamptic subjects were treated at the Boston Lying-In Hospital with "Veratrone" and magnesium sulphate. Two maternal deaths occurred, the patients being moribund on admission. In nearly half the cases the fœtus died. From 1873 to 1942, when the *régime* was instituted, the maternal mortality rate varied from 18·2% to 45·4%; but the author states that the results are now so much more satisfactory that the hospital will contrive to use the method until something better appears.

Neutral Diet and Hydration for Toxæmias of Late Pregnancy.

RUSSELL R. DE ALVAREZ (*American Journal of Obstetrics and Gynecology*, September, 1947) presents a method of treatment of the toxæmias of late pregnancy which has as its object an attempt to return involved tissues and organs to, and to maintain them at, as nearly physiological a state as possible. The general plan is based upon the utilization of a neutral diet, ammonium chloride, abundant fluids, bed rest, sedation, and treatment in hospital. The neutral diet is salt free and sodium poor, and consists of foods which yield an equal amount of acid ash and alkaline ash or an ash with no chemical reaction. Foods preserved in salt are prohibited—also the common heart-burn remedies which are rich in sodium bicarbonate. Sufficient water is required to carry off the metabolic end-products which the kidneys ordinarily excrete and fluids are forced to a level high enough to ensure an output equal to that of a normal individual, that is, two litres. Frequently a daily minimum intake of four litres is necessary to effect this result. If fluid is given intravenously, 5% glucose in distilled water is used to avoid the intake of sodium which produces œdema. Ammonium chloride is given to release the sodium ion from the tissues and the intercellular water retained by the sodium. Sedation is by means of phenobarbital. Convulsions are brought under control with "Sodium Pentothal". The author favours interruption of the pregnancy in all cases in which no improvement occurs after adequate medical treatment; medical induction and/or rupture of the membranes is preferred with normal spontaneous delivery (including "low forceps"). The indications for Cæsarean section should be essentially the same as when no toxæmia exists.

Dysmenorrhœa and Ovulation.

LOREN W. HAUS, JOSEPH W. GOLDZIEHER AND E. C. HAMBLEN (*American Journal of Obstetrics and Gynecology*, November, 1947) report the results of treatment of dysmenorrhœa with various dosage levels of œstrogen. They contend that one of the most striking characteristics of functional dysmenorrhœa is its invariable correlation with a progestational endometrium or an ovulatory type of basal temperature curve. Fifty-four patients were studied for a total of 228 cycles, and 82 biopsies and 11 basal temperature records were obtained. All anovulatory cycles were pain-free and all ovulatory cycles were accompanied by severe pain except in one case in which an early progestational endometrium was associated with no pain. Ovulation was inhibited by means of diethylstilbœstrol or œstrone sulphate ("Premarin") and the cycle dosage varied from 5 to 60 milligrammes of the former to 6·25 to 75 milligrammes of the latter. Dosage was controlled by means of endometrial biopsies or the basal body temperature graph, the optimum dosage being the minimum necessary to suppress ovulation. In general the chances of obtaining relief in any cycle or in any individual patient increase as the dose of œstrogen is increased. Treatment should not be considered a failure until it has been shown that the dosage was large enough to suppress ovulation, and in those patients in whom inadequate doses afford partial relief, one may expect total relief from adequate therapy. No gross menstrual disturbances followed the described schedule of œstrogen administration, although the dosage of some patients was relatively large. The authors conclude that the presence of progesterone or its physiological effects is a prerequisite for dysmenorrhœa, and although the symptoms are initiated by progesterone, the pathogenesis is unknown.

British Medical Association News.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioner has been released from full-time duty with His Majesty's Forces and has resumed civil practice as from the date mentioned:

Dr. Kevin W. Priddis, 217, Macquarie Street, Sydney (March 1, 1948).

Medical Societies.

MEDICAL WOMEN'S SOCIETY OF NEW SOUTH WALES.

A MEETING of the Medical Women's Society of New South Wales was held on October 10, 1947, at the Renwick Hospital for Infants, Summer Hill. The meeting took the form of a clinical demonstration arranged by the honorary medical staff of the hospital and conducted by Dr. Marjorie Dalgarno, Dr. Selina Puckey and Dr. Mona Nelson.

Duodenal Obstruction due to Peritoneal Bands.

The first baby shown had been aged three weeks when admitted to the Renwick Hospital on August 12, 1947, and had a history of vomiting since birth. The child had been admitted to the Royal Alexandra Hospital for Children at the age of two days and discharged on August 5, still vomiting, and passing green fæces frequently. Vomiting was not projectile, and the vomitus was bile-stained. On August 15 X-ray examination after a barium meal revealed apparent distension of the stomach. The head of the meal passed into the distended duodenal loop, but at the end of three hours none of the meal had reached the jejunum; this suggested probable atresia or severe obstruction due to narrowing in the duodeno-jejunal area. On August 16 the baby was dehydrated, and the condition was too poor for operation. By the continuous intravenous drip method 350 millilitres of saline and glucose solution were given. On August 17 operation was undertaken by Dr. G. B. Lowe. The stomach and duodenum were much distended, part of the duodenum was plum-coloured, and a number of peritoneal bands were present, causing obstruction at the duodeno-jejunal junction. The bands were cut and the abdomen was closed. Vomiting continued for a few days, but gradually ceased. The diet consisted of lactone syrup milk. On September 15 the baby was discharged from hospital, gaining in weight.

Pylorospasm.

A baby, aged six months, was shown with a history of vomiting since birth; weight gain had been satisfactory until the age of three months, when the vomiting became projectile in type. The child was admitted to the Royal Alexandra Hospital for Children and remained there seven weeks. The weight on admission to hospital was ten pounds six ounces and on discharge from hospital eleven pounds five ounces. For the month before the meeting the child had attended the clinic, and the weight was eleven pounds ten ounces, but vomiting was still present. The daily diet was one pint of cow's milk, two tablespoons and one teaspoon of farinaceous food and two tablespoons of lactone syrup. The diagnosis of pylorospasm was made at the Royal Alexandra Hospital for Children. On August 23, 1947, the baby was admitted to the Renwick Hospital for Infants; vomiting was present. Whilst the child was in hospital excessive secretion of mucus was noted, and possibly this was a factor contributing to the vomiting. "Eumydrin" was administered (half a tablet three times a day twenty minutes before food) from September 9 with good effect. The diet consisted of lactone syrup milk. On October 1 the child was discharged from hospital, having gained two and three-quarter pounds in weight. "Eumydrin" tablets were administered in the out-patient department, and the child did not vomit after discharge from hospital. It was explained that "Eumydrin" was given in this case in view of the possible pylorospasm and of the presence of excessive secretion of mucus.

Pyloric Stenosis following Pylorospasm.

The third patient, a baby, aged seven weeks, had begun to vomit five weeks previously; sometimes the vomiting was projectile. The birth weight was eight pounds eight ounces, and the weight at seven weeks was nine pounds three ounces in clothes. Peristaltic waves were seen, and projectile vomiting was occurring. The appearances seen in an X-ray film taken after a barium meal did not suggest pyloric stenosis. "Eumydrin" (half a tablet three times a day half an hour before food) was administered for the pylorospasm, and the diet consisted of lactone syrup milk. The child gained in weight and ceased vomiting for four weeks. Vomiting recommenced at the age of three months; it was projectile in character, and the child lost weight during the first week. A second X-ray examination after a barium meal showed the stomach to be full after three hours, and the diagnosis of pyloric stenosis was made. Rammstedt's operation for pyloric stenosis was carried out. It was pointed out that this was an atypical case of pyloric stenosis.

Empyema.

A child, aged two months, attended the clinic in July, 1947, on account of failure to gain in weight and of difficulty in sucking, and was referred to a general practitioner. The child was brought to the hospital later in the day as the appearances suggested impending convulsions. Coryza with cough had been present for three weeks. The baby was pale and underweight, the heart sounds were rapid and the breath sounds were diminished in the left lung. The percussion note was dull over the left lung anteriorly and posteriorly. The fauces were congested. X-ray examination revealed that the whole of the left side of the chest was radio-opaque and the cardiac shadow was displaced to the right; the appearances suggested a large collection of fluid. Penicillin was given, 5000 units intramuscularly every three hours, and sulphadiazine was administered. Chest aspiration was carried out on five occasions. On July 25, 60 millilitres of thick greenish pus were aspirated, on July 26 the amount was 40 millilitres, on July 27, 30 millilitres, on July 28, 15 millilitres and on July 30, 25 millilitres. *Staphylococcus aureus* was grown on culture. After aspiration on each occasion 10,000 units of penicillin were injected into the thoracic cavity. The diet consisted of a one in eight solution of condensed milk, and then of lactone syrup milk. The child was discharged from hospital after nineteen days, and after six weeks' attendance at the out-patient department the diet was changed from lactone syrup milk to one more suitable. The empyema apparently followed an attack of left lobar pneumonia. It was pointed out that the case was interesting, in so much as the child had no definite constitutional symptoms other than cough and feeding difficulties until the day of admission to hospital.

Unresolved Pneumonia, Probably Tuberculous.

A baby, aged five and a half months, had been admitted to the hospital on September 10, 1947, with a history of cough of three weeks' duration; the child perspired considerably and was feverish. On examination the temperature was found to be 100·8° F. and the child was flushed; the breath sounds were harsh and crepitations were present in both lungs, particularly the right. Neck rigidity was present and the knee jerks were exaggerated. Lumbar puncture was performed and eight millilitres of clear cerebro-spinal fluid under pressure were withdrawn; the fluid contained four cells per millilitre, and no growth of microorganisms was obtained on attempted culture. X-ray examination revealed that the upper two-thirds of the right side of the chest was radio-opaque; this was thought to be probably due to lobar pneumonia involving the whole of the upper lobe of the right lung. A small radio-translucent area suggested an area of localized pneumothorax. It was suggested that a further X-ray examination should be made in one week. Treatment was with sulphadiazine, and with penicillin given every three hours intravenously. The baby lost weight for three weeks, and then gained one and a half pounds over four weeks; intermittent pyrexia was present. On September 8 the tuberculin patch test produced a negative result, and on October 25 the result was positive. A Mantoux test on October 30 produced a positive result. The X-ray appearances in the chest remained unchanged. The diagnosis was made of unresolved pneumonia, probably tuberculous.

Double Right Ureter.

A baby, aged five and a half months, was shown who had a congenital abnormality of the urinary tract in the form of a double ureter on the right side. There was a history of pus in the urine since birth. The condition had been partly

relieved by treatment with sulphonamides and mandelic acid. The *Liquor Potassæ* test still produced a reaction. The child's nutrition was good and weight was being gained. A catheter specimen of urine was obtained in hospital; it was acid, and contained 500 to 600 pus cells, a few motile bacilli and some albumin. Culture produced a growth of *Bacillus coli communis*. On June 10, 1947, pyelography was carried out, dye being injected subcutaneously; no abnormality in the urinary tract was found. On June 18 a cystoscopic examination was made by Dr. Colin Edwards, and a retrograde pyelogram of the right ureter was obtained. On July 12 the child was admitted to hospital for sulphonamide therapy, which had a good effect; repeated examinations of the urine in the out-patient department showed that the infection was controlled. It was pointed out that, whilst the infection was under control, the child was to be examined by the urologist at the age of about three years unless incontinence of urine developed. If one of the double ureters opened below the urethral sphincter incontinence of urine would develop.

Osteomyelitis of the Femur.

The next patient shown was a child, aged two years and nine months. Although this patient clinically had osteomyelitis of the femur, no X-ray evidence was obtainable for twenty-four days. In spite of treatment with penicillin and sulphadiazine from the day of the child's admission to hospital, the infection continued to spread and involved the whole shaft of the femur. Considerable local œdema was present, but constitutional symptoms of the disease were never pronounced. Pathological fracture occurred in the distal third of the femur. It was pointed out that this was extremely rare in osteomyelitis, and callus formation was delayed for eight months. A large sequestrum involving two inches of the shaft was removed and after some weeks callus appeared. After that progress was steady, and several small sequestra had been removed. During one course of sulphadiazine treatment severe hæmaturia occurred. This cleared up when the drug was suspended and alkalis were administered. The child was discharged from hospital, walking, after thirteen months.

Obstructive Jaundice.

The eighth patient shown was a baby, aged five weeks, who had been jaundiced from birth; the fæces were white and the urine dark. On examination the child was seen to be jaundiced and the liver was palpable. Several conditions were considered in the differential diagnosis, the first being a blood dyscrasia, either an extreme degree of physiological jaundice or a pathological condition due to the Rh factor. The second condition considered was infection—(i) septicæmia (which usually arose from an infected umbilicus), (ii) any acute infection, or (iii) chronic infection, the most likely type being syphilitic. The third condition was a pathological condition of the liver or bile ducts (absence or atresia). A blood count gave the following information: the erythrocytes numbered 2,850,000 per cubic millimetre, the hæmoglobin value was 68% and the colour index was 1·2; the leucocytes numbered 27,000 per cubic millimetre, 42% being polymorphonuclear cells, 50% lymphocytes, 1% monocytes and 7% eosinophile cells. The erythrocytes were slightly irregular in size and shape. There was no evidence of any primary dyscrasia. Examination of the urine revealed no pus cells and no organisms, but a trace of albumin and some bile were present. The result of the Wassermann test was not available at the time of the meeting. The provisional diagnosis of a pathological condition of the bile ducts was made, and the child was to be submitted to laparotomy as soon as the general condition improved. The weight on admission to hospital was eight ounces below the birth weight. A diet of lactone syrup milk was given, and the weight increased by twelve ounces in ten days.

Pulmonary Tuberculosis.

The ninth patient had been admitted to hospital on September 18, 1946, suffering from pneumonia. The child had suffered from measles two months prior to the meeting; a cough had followed. X-ray examination revealed that the lower half of the right side of the chest was radio-opaque owing to pneumonia and interlobar effusion. The tuberculin patch test at the end of one month produced a positive result. The Mantoux test at the end of two months produced a positive response. The condition remained unchanged for two months, after which the child began to gain in weight, and examination of the chest revealed some signs of improvement, but some fullness was still present in the interlobar region. The patient was still under treatment.

Tuberculous Lobar Pneumonia.

The tenth case discussed was that of a girl, aged one year, suffering from tuberculous lobar pneumonia of the right lung with secondary bronchopneumonia in one lung. The child was radiologically examined as a contact of her father and mother, both of whom suffered from pulmonary tuberculosis. The lower two-thirds of the right lung was radio-opaque, probably owing to pneumonia involving the middle and lower lobes of the right lung. The cardiac shadow was not enlarged and the left lung was free from abnormality. The patient was admitted to hospital suffering from pneumonia. Progress X-ray films taken over the following four weeks showed that the condition was unchanged, and the appearances suggested tuberculosis. A tuberculin patch test at this stage produced a negative result. Six weeks after the child's admission to hospital X-ray examination revealed a large radio-translucent area in the lower portion of the right lung; this was considered to be due to a large area of localized pneumothorax, a lung abscess or a large tuberculous cavity. About this time the Mantoux test produced a positive result in fourteen hours. Six weeks later the child had a severe hæmoptysis. A further X-ray examination showed that the area of dulness in the right lung had extended to the level of the first rib, and a considerable increase in mottling had occurred in the left lung owing to the presence of bronchopneumonia, tuberculous in origin. The mottling increased during the next four weeks, and the child died five weeks after the hæmoptysis. The total loss of weight over five months was six pounds. The comment was made that this case illustrated tuberculosis commencing as lobar pneumonia which failed to clear up. The hæmoptysis and cavitation were unusual symptoms in infants.

Empyema followed by Pott's Disease.

A baby, aged ten and a half months, having been intermittently feverish for some days, was admitted to hospital on January 3, 1945, with the provisional diagnosis of pneumonia. An aunt had died of tuberculosis. On January 9 X-ray examination revealed an area of dulness extending along the lateral aspect of the right side of the chest, probably due to a collection of fluid. An area of dulness at the base of the right lung was thought to be due to basal pneumonia. The cardiac shadow was displaced to the left. On January 10, 90 millilitres of blood-stained fluid were aspirated from the chest; attempted culture of micro-organisms from the fluid produced no growth after twenty-four hours. On January 12 a further X-ray examination revealed that the effusion had diminished. A blood count revealed that the erythrocytes numbered 3,650,000 per cubic millimetre and the leucocytes 8000 per cubic millimetre; many granular cells were present. On January 24 X-ray examination showed the effusion to be still present. On January 31 some reduction had occurred in the mottling at the base of the right lung; dulness was present in the upper lobe of the right lung. On February 1 the temperature was normal and the patient was discharged from hospital.

On April 17 the child was readmitted to hospital; some weight had been lost and cervical adenitis was present. X-ray examination revealed an area of dulness towards the base of the right lung, possibly an interlobar effusion, and another area of dulness extending outwards from the right hilum. On April 22 the patient was discharged from hospital. In June a further X-ray examination revealed no change.

On October 9 the child was readmitted to hospital again with a history of cough of one week's duration. X-ray examination revealed an area of dulness to the right of the superior mediastinum and enlarged hilar glands. The lung fields were clear. In October the child was discharged from hospital. X-ray examination on April 24, 1946, showed that the enlarged hilar glands were still present, and the enlargement was thought to be possibly tuberculous in origin. Compression of the eleventh thoracic vertebra due to Pott's disease was now present. On July 28, 1946, destruction of the body of the eleventh thoracic vertebra had increased; this suggested that the tuberculous process was still active. On July 14, 1946, the X-ray appearances were much the same; there was a slight increase in the mottling in the upper lobe of the right lung. The patient was referred to the Royal Alexandra Hospital for Children for Pott's disease.

Discussion.

DR. WILLA NELSON expressed interest in the number of patients fed on lactone syrup milk, and asked what were its advantages. She also asked why, since lactone syrup milk appeared to be such a satisfactory diet for sick babies, it was not more frequently used for healthy babies.

Dr. S. Puckey, in reply, said that the advantages of lactone syrup milk were three: (i) the very fine curded state of the milk, which aided digestion of proteins; (ii) the satisfactory degree of acidity of the gastric contents which was maintained; (iii) the high calorific value of the milk (25 Calories per ounce). Disadvantages were expense and difficulties in preparation. Healthy babies did not need such highly specialized food.

DR. C. SAUNDERS discussed the patient suffering from pylorospasm treated with "Eumydrin" tablets. She referred to Dr. N. M. Jacoby's advocacy of "feed, volume reduction and atropine methylnitrate solution ('Eumydrin')" as medical treatment of pyloric stenosis in selected cases. Dr. Saunders was interested in the successful treatment of patients with "Eumydrin" in tablet form, which was much more convenient than Jacoby's fluid and could be given to out-patients.

DR. S. SIEDLECKY referred to the child with double ureter, and asked Dr. Puckey what the prognosis was.

Dr. Puckey, in reply, said that Dr. Colin Edwards did not consider interference advisable whilst infection could be controlled until the child reached the age of three years. In the event of one of the double ureters opening below the urethral sphincter, with the development of incontinence of urine, surgical interference would be considered.

DR. DORIS SELBY referred to the baby, aged five weeks, suffering from jaundice, and asked about the prognosis.

Dr. Puckey, in reply said that the provisional diagnosis was some pathological condition of the bile ducts, and laparotomy was proposed as soon as the patient's condition warranted operative interference; a blood transfusion would be given two or three days prior to operation.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the Institute of Medical and Veterinary Science, Adelaide, on March 28, 1947.

Scientific Research Affecting Agriculture in England and the United States.

Professor H. C. Trumble read a paper on some impressions of scientific research affecting agriculture in England and the United States of America. He said that biological research in Great Britain was largely financed and coordinated by the State. Three main government organizations established under the Lord President of the Privy Council were: Department of Scientific and Industrial Research, the Agricultural Research Council and the Medical Research Council. The relations between these three bodies were fairly close. Where agriculture touched industry, as in processing and storage, work concerning agricultural problems was undertaken by the Department of Scientific and Industrial Research. Where agriculture touched public health or medicine, work might be passed on to the Medical Research Council.

In addition to their relationships with government departments, the three research organizations kept in close contact with the universities; by making special research grants and facilitating cooperative effort, they assisted in stimulating the development of research and also helped to recruit young scientists for specific research bodies.

In soils work, modern geochemical methods were proving of considerable value. Quantitative spectroscopy, both X-ray and optical, was being generally employed. Development of the knowledge of crystal structure had permitted the determination of the atomic and ionic radii of many elements, thus enabling prediction of their occurrence to be made. The entry of ions or atoms into the three-dimensional network of the crystals was determined by their relative sizes and their order of entry by interatomic bondage forces.

Emphasis was being placed on increased fundamental work in plant physiology. The bearing of the original work on auxin in seedling oats on the use of growth substances for weedicides, the prevention of fruit drop, the parthenocarpic development of tomatoes and on fruit damage by frost at flowering were examples of valuable practical application of basic physiological work. Emphasis was tending to be placed on the importance of root excretions. Those from the potato tuber were known to assist the germination of the cyst of the potato eel worm, while cynamic acid excreted from the roots of guayule effectively inhibited the growth of guayule seedlings. The secretion of antibiotic substances by both higher and lower plants had given an impetus to investigations concerning soil microbiological competition. It was being found, however, that in many groups of soil microorganisms, taxonomy was still in a much confused state.

Work in biochemical genetics appeared to have been stimulated by the new conception of the gene-enzyme association, arising especially from the work of Beadle, now at the Californian Institute of Technology, and of Lindegren at Washington University, St. Louis. There was an active unit studying cell metabolism under the Medical Research Council in the Department of Biochemistry at the University of Sheffield with McIlwain. It appeared that certain bacterial reactions could be due to one or a few molecules of catalyst per bacterial cell. They included some stages in the synthesis, breakdown and conversion of vitamin-like substances. There was reciprocity of action between the sparsely distributed enzyme, presumably the gene, and plant synthesis.

A tribute was paid to the value of the work done by the Scientific Liaison offices during the war in both London and Washington. Specific examples of research in England and the United States were given.

University Intelligence.

DEMONSTRATION NIGHT IN THE PATHOLOGY DEPARTMENT OF THE UNIVERSITY OF MELBOURNE.

A DEMONSTRATION NIGHT was held on September 17, 1947, in the pathology department of the University of Melbourne. Nineteen exhibits were prepared by members of the staff and the research workers in the department. These covered a wide field of medicine and illustrated recent research work carried out in the medical school, the applications of the study of the pathology to clinical science in a number of conditions and the use of visual aids in medical teaching.

Cardiac Muscle Hypertrophy and Ventricular Aneurysms.

DR. T. E. LOWE demonstrated histograms showing the frequency distribution of the size of cardiac muscle fibres related to the individual muscles of the ventricle. These indicated that although in the normal and in the moderately hypertrophied heart the tension in each layer was probably uniform, this did not necessarily follow in grossly hypertrophied hearts. In the last-mentioned muscle hyperplasia was apparent. From these observations Dr. Lowe was able to evolve a mathematical approach to the formation of ventricular aneurysms of the heart, and to demonstrate that the type of ventricular wall deformity after infarction of individual muscles could be predicted in the examples so far considered.

Congenital Cardiac Anomalies.

DR. L. E. ROTHSTADT demonstrated several specimens showing various congenital anomalies of the heart. Most of the conditions were compatible with life beyond infancy. Reference was made to the importance of attempting to establish a clinical diagnosis of the exact lesion present, as in recent years surgical treatment had been used in certain anomalies of the heart.

Subacute Bacterial Endocarditis.

DR. BRUCE ROBINSON showed a number of specimens illustrating the pathological basis of the symptoms and signs of subacute bacterial endocarditis. Special reference was made to the dangers of tooth extraction in patients who had valvular abnormalities whether congenital or acquired, and the difficulty of obtaining a positive result on attempted culture of microorganisms from the blood was discussed. Dr. Robinson showed that while modern treatment might sterilize the valves, these became less competent through further scarring, though the ultimate cardiac failure from this cause might be preceded by uræmia from failure of kidneys damaged by emboli. For comparison specimens of the acute and chronic forms of endocarditis were displayed.

Degeneration of the Aortic Media (Erdheim's Medionecrosis).

DR. E. W. BATE presented an exhibit which demonstrated changes of muscle degeneration, elastic tissue loss, loose avascular scar formation and mucoid accumulation in the media of the aorta—the type of medionecrosis described by

Erdheim and Gsell. Specimens indicated the relationship of such medial degeneration to dissecting aneurysm and to direct "spontaneous" rupture of the aorta. The possibility of clinical diagnosis in about half the cases and its importance were stressed.

Studies in Tissue Culture.

DR. A. W. POUND presented some tissue cultures illustrating the behaviour of fibroblasts *in vitro* and the effects of tissue extracts on their growth. This "growth" was largely migration of cells, mitosis occurring when the cells reached a certain size. Functional activity of the cells to produce fibroglia and collagen fibres was shown to occur in regions where growth was less active. In certain circumstances in the medium change in morphology of fibroblasts to macrophages and round cells was illustrated. Although in many respects these phenomena *in vitro* differed from those in the intact animal, they were commonly seen in conditions of inflammation and repair, and were the physiological responses of the cells to certain changes in environment. Cinematographs of living fibroblasts illustrated the characteristic amœboid mode of movement of these cells and the behaviour of cells in mitosis.

The Early Development of Mammalian Embryos.

MR. W. B. MATHER, of the zoology school of the University of Melbourne, who was carrying out research into some aspects of experimental embryology, showed the following exhibits: (i) two-day chick blastoderms grown *in vitro* under aseptic conditions on clots formed from fowl plasma and nine-day chick embryo extract; (ii) unfertilized rabbit eggs collected from the oviducts after induced ovulation with the gonadotropic preparation "Synapoidin", together with the gross and microscopic appearance of the stimulated ovaries; (iii) films of the developing rabbit and monkey eggs prepared by the Wistar Institute.

Bone Marrow Culture.

DR. J. H. BOLTON pointed out that consideration of the chemistry of folic acid in relation to thymine metabolism suggested that the conversion of ribonucleic acid to desoxyribonucleic acid was a fundamental fault in pernicious anæmia. Observation of the growth of blood cells should therefore throw light on the mechanism of action of folic acid, and marrow culture would permit the economical testing of various degradation products of folic acid. The method of sternal marrow culture used by Israel was described and other results of preliminary experiments were discussed in which this technique was used. No significant changes occurred with folic acid aded to cells grown in placental serum, but a significant maturation of the erythroid series had been demonstrated by the addition of folic acid to cells grown in the patient's serum.

Carcinoma of the Thyreoid.

DR. DOUGLAS HICKS indicated the variety of tumours which might arise in the thyreoid gland. He said that differences in the histological appearances and in the behaviour of tumours of the thyreoid made it difficult to establish accurate criteria of malignancy. This led to considerable uncertainty as to the true incidence of definite cancer of the thyreoid, and also to difficulty in assessing the ultimate prognosis and the results of treatment. Surgery, combined with post-operative radiotherapy, seemed to produce the best results. It was hoped that treatment with radioactive iodine might be of value in some types of thyreoid cancer. Mounted specimens, together with microscopic preparations and photomicrographs, illustrated the different types of tumour.

"Serum Sickness" in Rabbits.

DR. MICHAEL KELLY presented a demonstration of the histological lesions seen in "serum sickness" in rabbits that were recorded by Rich (1942). These were illustrated by microphotographs of various tissues showing the changes brought about by large intravenous injections of horse serum. Dr. Kelly used these findings to draw attention to the similarity of the histological changes seen in serum sickness, *polyarteritis nodosa* and acute rheumatism noted by Bergstrand (1946).

Metropathia Hæmorrhagica.

DR. GRAHAM GODFREY dealt with the relationship between persistent follicular cysts of the ovary and certain cases of *metropathia hæmorrhagica* and showed endometrial biopsies from an illustrative case before and after resection of multiple small follicular cysts. Photomicrographs of the cysts were shown to demonstrate persistent granulosa cells, and clinical details were set out for correlation with the pathological findings.

Kidney Tumours.

DR. HOWARD H. EDDEY presented a demonstration showing the relationships of various kidney tumours and illustrated this with photomicrographs, specimens and pyelograms. He made the following points.

Renal tumours might arise in the renal substance or in the pelvis. Malignant tumours of the renal substance were: (*a*) adenocarcinoma—an adult tumour—and (*b*) embryoma (adenosarcoma)—an infantile tumour. Innocent tumours of the renal substance (adenoma, fibroma, lipoma) were of no practical significance. Tumours of the renal pelvis were (*a*) papilloma (innocent), and (*b*) papillary carcinoma and epidermoid carcinoma.

Pyelograms of adenocarcinomata showed variable distortion of the calyces and sometimes pelvic filling defects, the pelvis being invaded. In embryoma the pelvis was not invaded, but was, together with the calyces, distorted and obliterated. Pelvic tumours revealed their presence in the pyelogram by filling defects, and if the tumour had been at the pelvi-ureteral junction, by hydronephrosis. Stone was an accompaniment of epidermoid carcinoma in 50% of cases.

Segmental Nature of the Lungs.

DR. ALAN PENINGTON demonstrated the segmental character of the lungs by means of a brief review of the development of the bronchial tree and by dissections of normal bronchi. The nomenclature of the bronchi and of the broncho-pulmonary segments was denoted, and the segmental distribution of disease in the lungs was demonstrated by a specimen of lung infected with tuberculosis.

Glycogen Content of Liver Cells.

DR. J. W. PERRY presented a demonstration illustrating and extending the work of Chipps and Duff (1942). These authors had published a paper in 1942 on the glycogen infiltration of liver cell nuclei. Further observations had been made on material obtained by biopsies, by aspiration and by laparotomy of the liver and also on material obtained at autopsy. The results of these workers had been confirmed concerning the incidence of glycogen-containing nuclei in *diabetes mellitus*. A more delicate histochemical test with the Best's carmine stain "Feulgen Bauer" reaction had been applied and had proved that the intranuclear material was indeed a carbohydrate, and furthermore, it could be completely removed from liver sections by treating them with salivary amylase. Observations on post-mortem material showed that post-mortem autolysis was slower in the nucleus than in the cytoplasm. The nature of this abnormal distribution of glycogen was still in the realms of conjecture, especially in its relation to *diabetes mellitus*.

Intracranial Calcification.

DR. W. HAMILTON SMITH showed pathological specimens and X-ray films to illustrate the significance of intracranial calcification. He drew attention to calcification occurring in the pineal body, in the chorioid plexus and in the *dura mater*, especially the *falx cerebri*. Dr. Smith presented examples of cerebral tumours undergoing calcification, including gliomata, meningioma with psammoma bodies, and craniopharyngioma, and showed specimens and X-ray films of cerebral aneurysm, venous angioma, calcifying subdural hæmatoma and tuberculoma.

Bone Marrow Biopsy.

DR. JOHN COLEBATCH demonstrated the use of bone marrow biopsy by sternal puncture. With illustrations on a board, he gave some notes on the history, anatomy and physiology of the bone marrow, a comparison of the methods of needle puncture and surgical trephining, and an outline of the technique of sternal puncture. He described the normal marrow picture or myelogram, pointing out that variations occurred in childhood, and stressed the limitations of the method and the need for experience in interpreting the myelogram.

Dr. Colebatch described the sternal puncture findings and their clinical value in pernicious anæmia and in refractory or aplastic anæmias, in agranulocytosis and in leuchæmia (especially in atypical and aleuchæmic leuchæmia), in thrombocytopenic purpura, in skeletal neoplastic conditions, and in certain infections. He illustrated this description with microscopic preparations under oil and with numerous screen projections of transparencies in colour photographed from marrow smears.

Ulcerative Colitis.

DR. E. E. DUNLOP showed macroscopic specimens, photomicrographs and X-ray films illustrating specific and non-specific ulceration of the colon. Specific types of colon ulceration shown included bacillary and amœbic dysentery, tuberculosis and bilharzia. Non-specific ulcerative colitis included specimens from a subject of "simple ulcer of the colon" leading to sudden perforation and of Crohn's disease causing ileo-colitis and regional colitis, and material illustrating diffuse and segmental forms of the disease clinically termed chronic ulcerative colitis. Serious complications displayed included hæmorrhage, perforation, formation of sinuses and fistulæ, stenosis, polyarthritis, pseudopolyposis and the development of carcinoma. Treatment by ileostomy and colectomy was illustrated by pathological material and radiographs. It was suggested that non-specific ulcerative colitis was a clinical entity due to multiple causes rather than a distinct disease.

Intestinal Obstruction in Infants.

DR. A. MURRAY CLARKE demonstrated some causes of intestinal obstruction in infants, with special reference to atresia and stenosis and developmental errors resulting in an incompletely rotated caecum or an incompletely anchored mesentery. A brief summary of the embryology and possible anomalies was given. It was pointed out that surgery in many of these cases could be successful only if the underlying pathology was clearly understood and if one adopted the principles learnt from the study of pyloric stenosis, a high intestinal obstruction, whose mortality rate had been reduced from 15% to 2·8%. For example, in intestinal obstruction in infants an ileostomy was invariably unsuccessful, as it was impossible to combat the extreme dehydration by any means; and in volvulus of the small intestine, unless it was understood that, for a very good pathological reason, duodenal stenosis was often associated, surgical intervention would fail.

Intestinal Lipodystrophy.

DR. A. G. LIDDELOW showed a subject of intestinal lipodystrophy (Whipple's disease), a bizarre pathological condition causing chronic fatal fatty diarrhœa. In this instance there was the usual diffuse deposition of fat in the intestinal mucosa and lymphatics and mesenteric lymph nodes, with enlargement and fibrosis of those nodes. Polyserositis was manifested by adhesive pericarditis, pleural adhesions and patchy chronic peritonitis.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 39 and 42, of March 4 and 11, 1948.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Volunteer Reserve.

To be Surgeon Lieutenants.—Brian Oxenham, 17th December, 1946, seniority in rank, 12th September, 1942; Archibald Gordon Murray, 19th September, 1947, seniority in rank, 8th September, 1945.

AUSTRALIAN MILITARY FORCES.

Interim Army.

Australian Army Medical Corps.

SX700021 Captain P. G. Jay is appointed from the Reserve of Officers (Australian Army Medical Corps) (4th Military District), 13th November, 1947.

16th Camp Hospital.—SX700022 Honorary Captain R. A. Kenihan is appointed from the Reserve of Officers (Australian Army Medical Corps) (4th Military District) and to be Captain, 15th January, 1948.

20th Field Ambulance: To be Temporary Major, 25th November, 1947.—SX34533 Captain R. S. Colton.

Reserve of Officers.

Australian Army Medical Corps.

The following officers are transferred to the Reserve of Officers of the Military Districts shown on the dates indicated. Officers holding temporary rank relinquish such temporary rank with effect from the date of transfer to the Reserve of Officers:

NX207585 Captain J. M. Ellis (2nd Military District), 13th January, 1948.

70th Camp Hospital.—NX202334 Captain A. W. Middleton (2nd Military District), 25th November, 1947.

90th Camp Hospital.—NX208061 Captain G. C. Scott (2nd Military District), 23rd December, 1947.

Reserve Citizen Military Forces.

Australian Army Medical Corps.

1st Military District.—The notification respecting the retirement of Captain C. T. Petherbridge which appeared in Executive Minute No. 188 of 1947, promulgated in *Commonwealth Gazette* No. 241 of 1947, is withdrawn.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

WEEK-END COURSE AT KATOOMBA.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course will be held at Katoomba in conjunction with the Blue Mountains Medical Association in the lecture room of the Blue Mountains District and Anzac Memorial Hospital on Saturday and Sunday, April 10 and 11, 1948. The programme is as follows.

Saturday, April 10, 1948.—2 p.m.: Registration. 2.30 p.m.: "Epilepsy", Dr. Eric Susman. 4 p.m.: "Common Head Injuries", Dr. Douglas Miller.

Sunday, April 11, 1948.—10 a.m.: "Obstetric Views from Abroad", Professor Bruce T. Mayes. 11.30 a.m.: "Some Aspects of Encephalitis", Dr. Eric Susman. 2.30 p.m.: "Relief of Pain", Dr. Douglas Miller. 4 p.m.: "Progress in the Management of: (*a*) Breech Delivery, (*b*) Pre-eclamptic Toxæmia and Hypertension, (*c*) Cæsarean Section", Professor Bruce T. Mayes.

The fee for the course will be £1 1s. Those wishing to attend are requested to notify Dr. Nicholas Larkins, Honorary Secretary, Blue Mountains Medical Association, as soon as possible.

Correspondence.

ARTHROGENIC SCIATICA AND DISK PROLAPSE.

SIR: In your issue of December 13, 1947 (page 711), Mr. E. F. West, of Adelaide, has made a valuable contribution to our understanding of sciatic pain by stressing that limb pain may arise from lumbar joint derangement independent of disk prolapse.

It has hitherto been generally accepted that in herniation of the *nucleus pulposus*, limb pain is the result of spinal nerve root compression. While this may be a factor there is good evidence that other influences are involved. As was shown by Lewis and Pochin, in nerve paralysis from pressure touch sense fails early, motor power fails next, "fast" pain later, and delayed pain last. Before pain sensation is lost transitory hyperalgesia may occur, so that the earliest evidences of root compression are alternating periods of numbness and cutaneous tingling. Pain, such as that of the sciatic syndrome, does not occur. Further, as was shown by Lewis and Kellgren, painful stimulation of a deep somatic structure such as an interspinous ligament, by injection of hypertonic saline, results in pain throughout the corresponding sclerotome with accompanying muscle spasm. From personal experience and self-experiment we can aver that the pain produced by so stimulating the fifth lumbar interspinous ligament is indistinguishable from that occurring spontaneously in an L5–S1 disk lesion.

Now, like the lumbo-sacral arthritis which Mr. West describes, herniation of the *nucleus pulposus* is primarily a joint lesion. It causes strain on surrounding ligamentous structures and may secondarily give rise to spinal or nerve root compression. Such a joint lesion is analogous to the experimental irritation of deep somatic structures and may reasonably be expected to give rise to pain in the corres-

ponding sclerotoma, irrespective of whether or not nerve pressure occurs. In fact a large proportion of patients in whom disk prolapse is subsequently found at operation never present any acceptable evidence of nerve pressure, such as muscle weakness, anæsthesia or loss of deep reflexes. We hold that pain by itself cannot be regarded as evidence of root compression, and your contributor's observations bear this out.

If we accept this thesis the reason for disappointing operative results becomes clearer. Removal of the disk will reduce mechanical disturbance of the surrounding tissues and thereby diminish the irritation which gives rise to segmental pain, but it will not invariably abolish this pain, because in some cases the joint remains grossly deranged, to act as a constant source of painful stimulation. On this basis the need for spinal fusion in such cases becomes apparent. This, however, is a severe and time-consuming procedure, wider application of which would demand considerable extension of available orthopædic facilities. In time, an encouraging proportion of patients recover without operation, and this provides a valid argument for enthusiastic persistence with conservative methods. In particular we would decry the practice of exploring intervertebral disk spaces when no prolapse is apparent at operation. Such a procedure can only give rise to an unstable joint and must result in removal of many healthy disks.

Two of our recent observations we believe to be relevant to the present argument. (i) Application of a short plaster corset, even without preliminary extension, may very rapidly relieve pain, although anæsthesia persists and may actually increase as in one case under our observation. (ii) We have found that in many cases of disk prolapse injection of 1% novocaine into the corresponding interspinous ligament will produce relief from pain, lasting from a few hours to three weeks. Clearly, in neither instance is benefit due to relief from nerve root compression; here a purely mechanical interpretation of the functions of the central nervous system can only result in confusion.

We are at present engaged in an investigation of sciatic pain mechanisms and hope to communicate our findings in due course.

Yours, etc.,

J. DONALDSON CRAIG, M.D. (Lond.), M.R.C.P. (Lond.), M.R.C.S. (Eng.), Research Fellow in Medicine, St. Mary's Hospital, London.
A. W. LIPMANN KESSEL, F.R.C.S. (Eng.), Orthopædic Registrar, St. Mary's Hospital, London.

72, Wesbourne Terrace,
London, W.2,
March 3, 1948.

Bibliography.

J. D. Craig and A. W. L. Kessel: "Limb Pain in Intervertebral Disc Lesions" (in the press).

J. H. Kellgren: "On the Distribution of Pain Arising from Deep Somatic Structures with Charts of Segmental Pain Areas", *Clinical Science Incorporating Heart*, Volume IV, 1939, page 35.

J. H. Kellgren and T. Lewis: "Observations Relating to Referred Pain, Viscero-motor Reflexes and other Associated Phenomena", *Clinical Science Incorporating Heart*, Volume IV, 1939, page 47.

T. Lewis and E. E. Pochin: "Effects of Asphyxia and Pressure on Sensory Nerves of Man", *Clinical Science Incorporating Heart*, Volume III, 1938, page 141.

THE IMPORTATION OF BOOKS.

SIR: I recently read in THE MEDICAL JOURNAL OF AUSTRALIA the review of "Cancer: Diagnosis, Treatment and Prognosis", by Ackerman and Regato. The review was so good that I sent to a bookseller for the book. I was astonished and shocked to receive in reply the following circular letter.

> On a check through our records today we find that we have your name recorded for a book that we did not have in stock at the time of your order and which we were to supply as soon as we received copies.
>
> Due to the dollar shortage, the Commonwealth Government suspended the issuing of import licences last August and we have therefore been unable to order any American books from that time.
>
> As the result of a recent Commonwealth Government instruction, booksellers are now only permitted to import prescribed textbooks from dollar areas up to ten (10) per cent. of the total value of books imported by the bookseller for the year ended 30th June, 1947.
>
> It will be appreciated that the supply of textbooks we will be allowed to order from U.S.A. will be hopelessly inadequate to meet the demands of medical and dental students.
>
> Apart from this quota of textbooks, no other American books may be imported. This restriction applies even to books recommended for study for higher degrees. In view of the regulations we are reluctantly compelled to cancel the order you placed with us.
>
> Your name will be kept on our records, and if and when we are again permitted to order the book you require we will notify you.
>
> The above remarks do not apply to journals and magazines. These may still be imported.

The result of my not being able to get this book is that I do not know as much about cancer as I would do if I could get it. This means, of course, that those of my patients who suffer from cancer will be less well treated than they should be. There are times when we just have to accept the inevitable. It is very difficult to understand, however, why essential textbooks should be kept out and popular magazines such as the New York *Life, Woman's Home Journal, et cetera*, should be allowed to come in.

Yours, etc.,

C. CRAIG,
Surgeon Superintendent.

General Hospital,
Launceston,
Tasmania.
March 11, 1948.

EGG LAYING BY HENS.

SIR: I was interested to read in a recent scientific article in the journal that a hen will consistently lay one egg a day.

This reminded me of the experience of a colleague who was interested in a poultry farm. He recognized the above-mentioned fact and decided to profit by it. He found that during the long days of summer, by shutting his hens up in a dark place for a couple of hours in the middle of the day the result was that his hens laid twice in the twenty-four hours, a very profitable procedure.

However, on visiting his poultry farm early one morning after a very severe thunderstorm he was distressed to find that his hens were all dead.

The reason was obvious: there was ample evidence to show that the unfortunate hens had endeavoured to keep pace with the lightning, but the strain had been too much and they perished from exhaustion.

Yours, etc.,

ERNEST CULPIN.

Ballow Chambers,
Wickham Terrace,
Brisbane, B.17.
March 8, 1948.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Callander, Robert James, M.B., B.S., 1945 (Univ. Melbourne), Broken Hill and District Hospital, Broken Hill.

Caplan, David, M.R.C.S. (England), L.R.C.P. (London), 1940, D.O.M.S., 1942, 2, Vernon Avenue, Eastlakes.

Gibson, Andrew Gavan, M.B., B.S., 1942 (Univ. Melbourne), Barham.

Godby, Norman Russell, M.B., B.S., 1939 (Univ. Melbourne), 113 R.G.H., Concord.

Lloyd, Olive Tyndale, M.R.C.S. (England), L.R.C.P. (London), 1928, East Gresford.

Malley, Martin Joseph, M.R.C.S. (England), L.R.C.P. (London), 1928, c.o. Union Bank of Australia, Haymarket Branch, Sydney.

Sendak, Moses, M.R.C.S. (England), L.R.C.P. (London), 1926, M.B., B.S., 1927, M.D., 1937 (Univ. London), D.P.H. (England), 1937, c.o. the National Bank of Australasia, 342, George Street, Sydney.

The following additional qualifications have been registered:

Gray, Eva, 44, Bellevue Road, Bellevue Hill (registered in accordance with the provisions of Section 17 (*a*)

of the *Medical Practitioners Act*, 1938-1939), M.B., B.S., 1947 (Univ. Sydney).

Bolliger, Walter, 300, Sailor Bay Road, Northbridge (M.B., B.S., 1945, Univ. Sydney), D.P.H., 1947 (Univ. Sydney).

Carter, Leonard, 23, Wunulla Road, Point Piper (M.B., B.S., 1945, Univ. Sydney), D.P.H., 1947 (Univ. Sydney).

Cunningham, Norman Charles, 185, Macquarie Street, Sydney (M.B., B.S., 1930, Univ. Sydney, M.R.A.C.P., 1938), F.R.A.C.P., 1947.

Dowling, John Laidley, 1, Rose Bay Avenue, Bellevue Hill (M.B., B.S., 1937, Univ. Sydney), M.S., 1946 (Univ. Sydney), F.R.C.S., 1947 (England).

Maitland, Herbert Lethington Chisholm, 147, Macquarie Street, Sydney (M.B., Ch.M., 1923, Univ. Sydney), F.R.C.S. (England), 1947.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Penington, Alan Harry, M.B., B.S., 1933 (Univ. Melbourne), M.D., 1936 (Univ. Melbourne), Royal Hobart Hospital, Hobart.

Bruce, Albert Hamilton, M.R.C.S. (England), L.R.C.P. (London), 1927, F.R.F.P.S., 1947 (Glasgow), Tasmania.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Brandt, Donald Sutherland, M.B., B.S., 1939 (Univ. Sydney), 5, Vernon Street, Strathfield.

Duffey, Brian Thomas, M.B., B.S., 1946 (Univ. Sydney), St. Joseph's Hospital, Auburn.

Jones, Margaret Mary, provisional registration, 1947 (Univ. Sydney), Lithgow District Hospital, Lithgow.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association:

Close, Rosemary Jocelyn, M.B., B.S., 1947 (Univ. Adelaide), 424, Gilles Street, Adelaide.

Kneebone, John Keith, M.B., B.S., 1947 (Univ. Adelaide), 62, Anzac Highway, Everard Park, South Australia.

Maddison, Thomas Glover, M.B., B.S., 1946 (Univ. Adelaide), Laught Avenue, Forestville, South Australia.

Camens, Ivan Maurice Henry, M.B., B.S., 1947 (Univ. Adelaide), Pitcairns, Cross Roads, Glen Osmond, South Australia.

Medical Appointments.

Dr. Mary Lane has been appointed a member of the Council of Public Education representing the Education Department of Victoria.

Dr. J. H. Nicholls has been appointed honorary medical officer of the Mount Gambier Hospital, South Australia.

Dr. R. B. Austin has been appointed a member of the Advisory Council of the New England University College at Armidale, New South Wales, in pursuance of the provisions of Section 43 (3) of the *University and University Colleges Act*, 1900-1937, of New South Wales.

Books Received.

"The Electron Microscope", by V. E. Cosslett, Ph.D., M.Sc., M.A., F.Inst.P.; 1947. London: Sigma Books, Limited. Sydney: Walter Standish and Sons. 7¾" × 5", pp. 136, with many illustrations. Price: 7s. 6d.

"Victory Over Pain: A History of Anesthesia", by Victor Robinson, M.D.; 1947. London: Sigma Books, Limited. Sydney: Walter Standish and Sons. 8¼" × 5½", pp. 352, with illustrations. Price: 16s.

"Endocrine Therapy in General Practice", by Elmer L. Sevringhaus, M.D., F.A.C.P.; Sixth Edition; 1948. Chicago: The Year Books Publishers, Incorporated. 8" × 5¼", pp. 264, with many illustrations. Price: $4.00.

"Clinical Methods in Surgery", by K. Das, M.B. (Cal.), F.R.C.S. (England and Edinburgh); 1947. Calcutta: Hilton and Company. Sydney: Angus and Robertson, Limited. 9¾" × 7", pp. 250, with many illustrations.

Diary for the Month.

MARCH 30.—New South Wales Branch, B.M.A.: Council Meeting (election of officers).

APRIL 1.—South Australian Branch, B.M.A.: Council Meeting.

APRIL 2.—Queensland Branch, B.M.A.: Branch Meeting.

APRIL 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.

APRIL 7.—Western Australian Branch, B.M.A.: Council Meeting.

APRIL 7.—Victorian Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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